ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 49 MARCH, 1940 No. 1

I

A TALK TO HOUSE OFFICERS AND THE PHARYNGO-MAXILLARY APPROACH TO THE TIP OF THE TEMPORAL BONE*

HARRIS P. MOSHER, M.D.

BOSTON

PART I

The first part of the following article is a talk to House Officers which I gave this year. Each member of the staff is scheduled for at least one lecture a season as part of the instruction of the House Staff. Usually the subjects are of a formal didactic character, their purpose being to cover the fundamental subjects of otolaryngology. Strange as it may seem, I was a bit tired of talking on the esophagus or osteomyelitis of the frontal bone, and gave instead of either topic a somewhat cursory and at times theoretical talk revolving about the general subject of petrositis.

The talk as given to the House Staff has been extended a little by the addition of the report of a recent case of petrositis where the infection sought a means of escape by way of the pharynx, and the petrous tip was approached through the pharyngomaxillary fossa by the submaxillary route.

Last April, at the Medical School, I gave myself a lonely refresher course in the anatomy of the temporal bone, and I needed it.

*All the illustrations are by the writer.

Fig. 1. The illustration shows a temporal bone cut book fashion. The bone is cut as indicated in the drawing. The sectioning begins at the tip of the temporal bone and continues through to the limit of the mastoid process. The sections or leaves can be made of any thickness, usually a strong quarter of an inch. The external soft tissue is not cut through so that the specimen holds together and can be opened like a book. It has a great advantage over serial detached specimens for studying and teaching.

I was interested to find out how the cellular development of the mastoid ran in a series of dissecting room heads. I took 83 mastoids. All were adult heads and most of the specimens were half heads. In addition, I examined, with the help of Dr. Ogden and Dr. Kos, 88 cleaned skulls from the teaching collection of the Anatomy Department. Dr. McCall, Dr. Kos and Dr. Ogden cut the wet heads for me.

What I have to discuss in part are certain anatomical points shown by the specimens. Beside points in the applied anatomy of the temporal bone, I shall take up some clinical applications, namely, the continuity of the marrow spaces or cells of the body of the sphenoid with the marrow spaces of the tip of the temporal bone; osteomyelitis of the dorsum sellae and the body of the sphenoid, and retro-orbital pain not due to petrositis acute or chronic, but caused, I feel, by infection from the posterior ethmoidal cells, but more especially from the sphenoidal sinus.

This statement of the topics of the paper gives also the order of the paper.

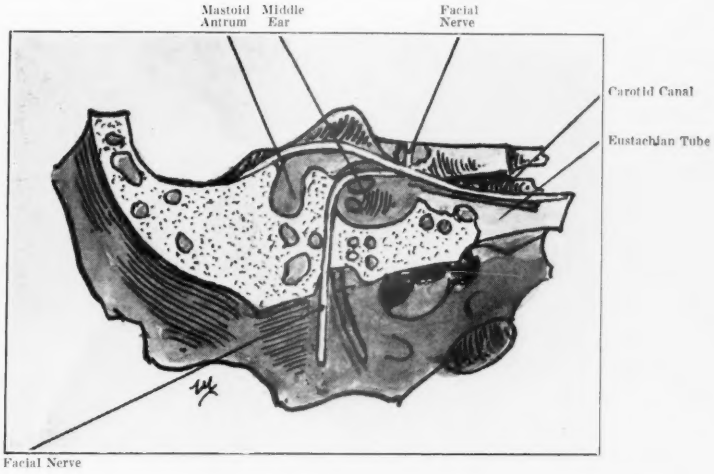


Fig. 2. The illustration is to show normal anatomy. It is drawn from a temporal bone which has few cells. From the specimen it is easy to locate the main regions from which the cells of the temporal bone spring, namely, the mastoid antrum, the infra-cochlear space and about the eustachian tube. With each new investigator the classification of the cells becomes more elaborate and complicated. From the practical standpoint I prefer the simple classification just given. It is taken from Wilson.

What I have to give may appear a bit tenuous in theory. At least Dr. MacMillan intimated as much. For instance, in talking with him about my conception of osteomyelitis of the dorsum sellae, he said that it was so common as to be of no significance. My answer is that if you can find the cause of the common you often find the cause of the less common. The common man is still the hope of the world. The uncommon man is making a horrible mess of it. This, of course, is not a personal reflection on Dr. MacMillan.

Quite a few years ago Eagleton advised us to watch the sphenoidal sinus. He felt that in cases of pneumococcic meningitis Type III the focus was often to be found in the sphenoidal sinus. He also stoutly maintained that in petrositis the pathological lesion was usually an osteomyelitis of the marrow spaces of the tip, rather than always an osteitis of the pneumatic cells. I have a feeling that in many cases he is right.

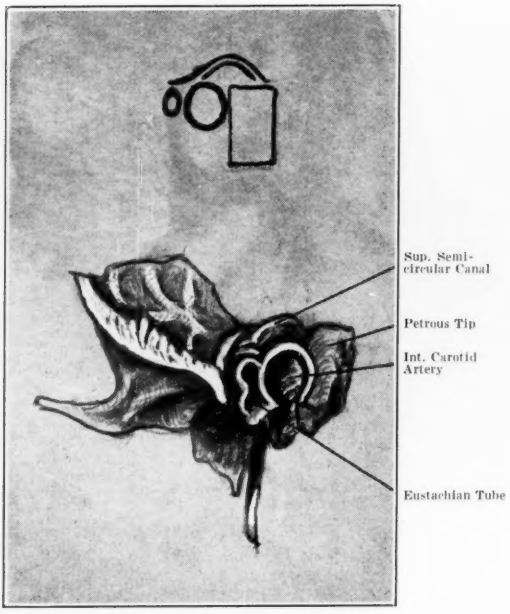


Fig. 3. Natural size. The cut shows the tip of the disarticulated temporal bone. The transverse measurement of the cellular part of the tip is usually a strong quarter of an inch and the height a strong half inch. The upper figure of the cut is a diagrammatic representation of the structures found in the petrous tip. The markers of the lower drawing identify them.

MATERIAL

As I just stated, I examined 83 mastoids from dissecting room material. All were wet specimens and all were from adults. The great majority were full half heads, the smaller number temporal bones only (Fig. 1).

Twenty-five specimens were cut vertically and in book form, and 60 were cut horizontally through the internal auditory meatus. Of the 83 specimens 36 were sclerotic and 47 pneumatic, a majority of 11 in favor of the pneumatic type.

Zygomatic cells were present in 15 specimens but in only two were they of any considerable size.

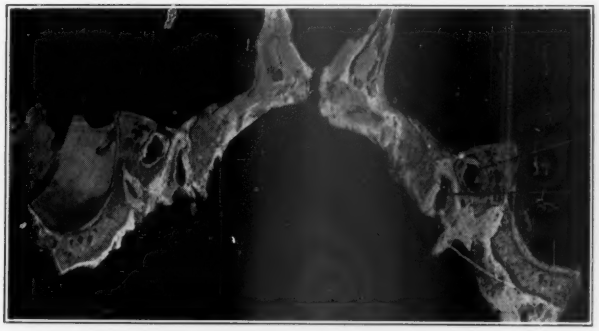


Fig. 4. Illustration showing the relation of the eustachian tube to the internal carotid artery.

Tip cells were present in 40 of the 83 specimens. These tended to cluster about the carotid artery.

Eighty-eight cleaned skulls also were examined.

THE CLASSIFICATION OF THE CELLS

Lindsay states that in patients over three years of age the petrous apex is pneumatized in 21 per cent. He has a rather elaborate classification of cells—namely, superior cells, postero-medial (these are subdivided), and peri-tubal. The superior cells lie between the arcuate eminence and the internal auditory meatus. Such cells are found in 27 per cent of the cases where the tip is pneumatized. Where these cells are present, in 19 per cent they are walled off from the tip. (See Fig. 2.)

The simplest classification of cells is that given by Wilson; namely, superior cells rising from the antrum of the mastoid and inferior or tubal cells which run below the cochlea and may reach the tip on any side of the eustachian tube, at times even making a spiral about the tube. (See Fig. 3.)

If you examine an exenterated mastoid you will see that the posterior cells and the infra-labyrinthine cells are in relation with the lateral sinus, and the inferior petrosal sinus is in relation to the tip of the petrous portion of the temporal bone so that infection can reach the tip by way of the lateral sinus and the inferior petrosal sinus.

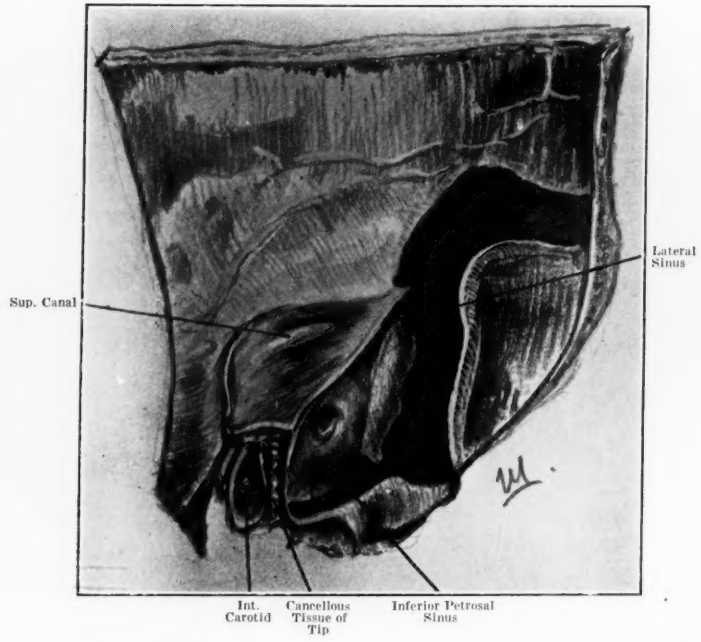


Fig. 5. In the specimen the petrous pyramid has been disarticulated from the sphenoid in order to show the tip. It shows the relation of the inferior petrosal sinus to the petrous tip. This runs practically the whole length of the under surface of the tip.

This occurs either by the infected blood stream or by infection transmitted along the walls of the two blood sinuses. In this way, infection can circumvent the labyrinth and reach the marrow spaces of the tip, and it is not necessary, therefore, to have a continuous pathway of pneumatic cells from the mastoid proper to the petrous tip for infection of the tip to occur, whether the tip is made of pneumatic cells or marrow cells.

THE PERIOSTEAL UNION BETWEEN THE TIP OF THE TEMPORAL BONE AND THE BODY OF THE SPHENOID

The junction between the petrous tip and the basilar process of the occipital bone occurs just lateral to the vertical part of the internal carotid artery. The periosteum joining the two is often

seen in the sections which I studied. However, it does not completely wall off the tip from the basilar process, because the marrow spaces of the basilar process in most of my 83 specimens were continuous into the petrous tip without interruption.

By searching along the line of periosteum, when it happens to be visible, a sharp searcher will often drop into sizable cavities somewhere along the course of the suture line. Such a cavity is not uncommon in the petrous tip lateral to the carotid artery. These cavities could easily show in x-ray films as sizable tip cells. (See Fig. 6.)

In 88 cleaned skulls, in 12 both tips were fused with the basilar process of the sphenoid, and six were fused on one side only, fusion taking place on one or both sides in about 20 per cent.

It is old knowledge how close the extensions of a large sphenoidal sinus can be to the tip of the petrous, to the body of the sphenoid, to the cavernous sinus, and to the gasserian ganglion, especially its first branch, the ophthalmic. It is close, also, to the sixth nerve. (See Fig. 7.)

There are two more applied points in anatomy which I might mention; namely, the first part of the facial nerve as it runs from the internal auditory meatus to the knee or geniculate ganglion is almost straight. In order to miss the nerve the operator should be a strong quarter of an inch beyond the superior semicircular canal before attempting to pierce the cortex of the petrous tip. The examination of anatomical specimens makes one feel that the surest anatomical route from the middle ear to the petrous tip is along the internal carotid artery or the route of Remadier. This presupposes that in any given case, there is no fistula in the middle ear to follow (Fig. 8).

On the examination of the specimens, the first and perhaps the most striking observation was the fact the capsule of the inner ear seemed to be suspended in surrounding cells, the mastoid cells behind, the marrow spaces in front or internally. This is shown beautifully also in the x-ray, but it is satisfactory to see it in the actual specimens because the x-ray remains so uncanny to many of us that there is still an air of necromancy about it. In none of the 83 specimens was there a macroscopic cellular route to the tip superiorly, that is, above the superior semicircular canal, and in only two or three was there a cellular route to the tip below the capsule (Figs. 9 and 10).

The second striking feature of the sections of the half heads was the apparent continuity of the marrow spaces of the basilar process of the sphenoid, with similar marrow spaces running without

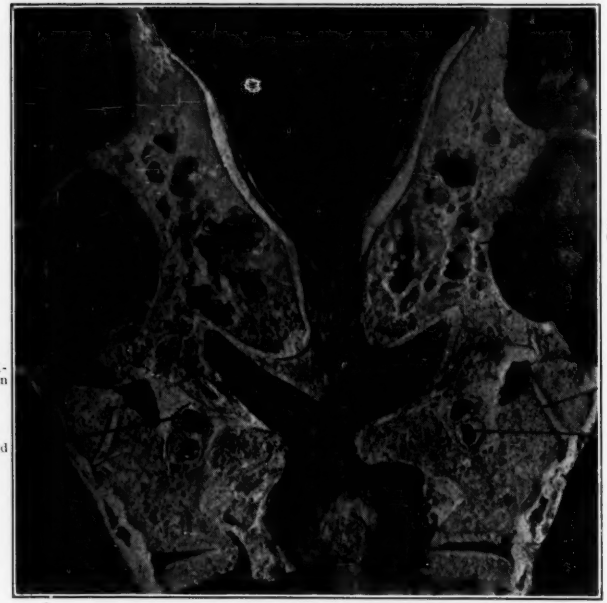


Fig. 6. The illustration shows a markedly pneumatic temporal bone. In places the periosteal junction between the temporal bone and the sphenoid shows in the cut. There is a large bone cavity above the internal carotid and another in connection with the periosteal junction. Large spaces are often found in this situation. In the great majority of specimens of the temporal bone cut horizontally through the internal auditory meatus, no periosteal separation between the sphenoid and the tip of the temporal bone was seen. In other words, the marrow spaces of the sphenoid were continuous without break into the tip of the temporal bone.

a break into the petrous tip, often reaching nearly to the internal auditory canal. Many writers maintain that there is a histological break between the two types of bone. No break is apparent to the naked eye. The continuity is so strikingly complete that I find it hard to believe that the histological break is of any clinical importance. I do not believe that infection would find any difficulty traveling from the air cells to the marrow spaces. We have in an extensively pneumatized mastoid a sufficient explanation for infection working from without inward and involving the tip, the middle ear being the source of the infection. For the tip to be infected from within, as it were, the infection can come from the sphenoidal sinus. The anatomical relationship of the sphenoidal sinus to the marrow spaces of the basilar process and the marrow spaces of the petrous tip is well known, as I just said, to be extremely intimate at times. I feel that infection can originate in the sphenoidal sinus and extend to the petrous tip, resulting in an osteomyelitis of the myeloid tissue of the petrous tip and of the basilar process (Fig. 11).

Wilson maintains—I am repeating—that there is no connection between the marrow cells of the tip and the pneumatic cells when they happen to be present together. Lindsay feels much the same way, but he states that there is always a protective fibrosis in the marrow cells when the neighboring pneumatic cells are infected. This fibrosis is an early reaction of the marrow cells to infection and means that they are becoming involved. It is beautifully shown in osteomyelitis of the frontal bone. In one instance in a petrositis specimen, Lindsay found actual abscess formation in the marrow cells adjacent to infected air cells (Fig. 12).

On examining the marrow spaces of the body of the sphenoid, large spaces—large enough to simulate pneumatic spaces—were found to be frequent, and such spaces were common about the internal carotid artery where it turns forward into the cavernous sinus. Such large cells, especially those about the carotid artery, look like catch basins ready to pick up infection. It is easy to see that if the cells or marrow spaces about the carotid became infected, infection could readily extend to the tip of the temporal bone (Fig. 13).

One of the first specimens examined showed two large tip cells. This specimen gave a chance to see how close tip cells could come to the sixth nerve. Both of the two cells practically touched it. On freeing up the Gasserian ganglion, the relation of its first division—the ophthalmic—to the cavernous sinus and to the side of the sphenoidal sinus was clearly shown. The ganglion lay on the lateral side of each of these structures (see Fig. 6). Infection from either one

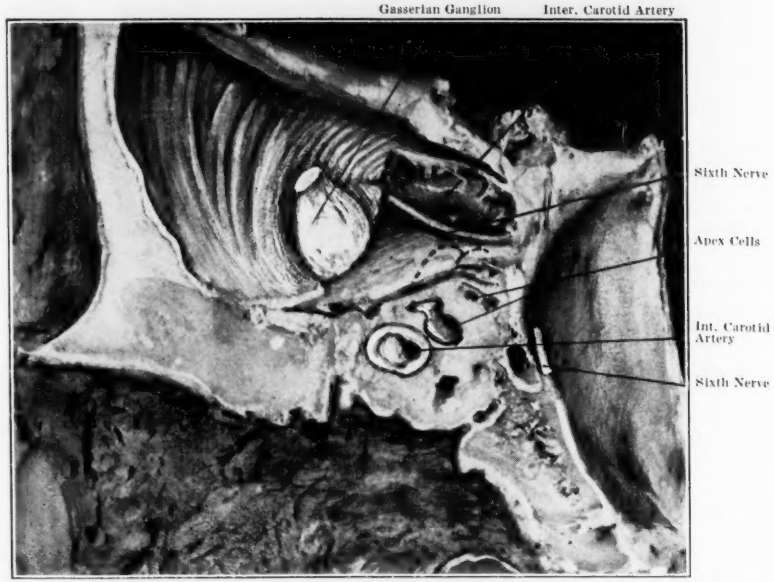


Fig. 7. The illustration shows the standard relationships of the Gasserian Ganglion. The ganglion has been turned out of its bed. When it is back in place the nearness of the first division (opththalmic) to the cavernous sinus and the sixth nerve and the internal carotid artery contained in it are strikingly shown. In the specimen there are two large tip cells. Each practically touches the sixth nerve. The uphill and subdural course of the first part of the sixth nerve come out clearly.

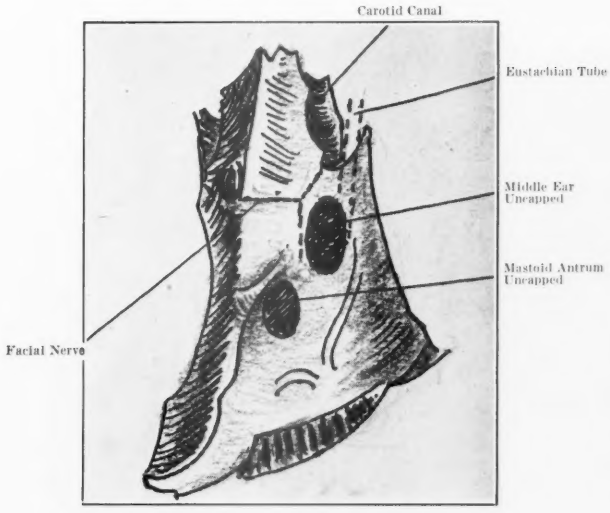


Fig. 8. The illustration is from a drawing showing certain points in the applied anatomy of the temporal bone.

could not fail to reach it. Retro-orbital or ganglion pain could be easily explained by these relationships. The nearness of the sixth nerve to the cavernous sinus and to the tip cells of the petrous portion of the temporal bone plus its imprisoned dural course, readily accounts for its paralysis by neighboring swelling arising from infection in one or both of the places just mentioned.

BONE OPENINGS FOR THE PASSAGE OF VEINS

The posterior surface of the basilar process of the sphenoid often shows pits for the passage of veins. In 80 of the 88 cleaned skulls these pits were found. I have found these large venous bone openings in the bottom of the sella tursica and on the side of the sphenoidal sinus. The books on anatomy stress the point that the roof and the sides of the body of the sphenoidal sinus are cribriform for the passage of veins (Figs. 14 and 15).

The under surface of the body of the sphenoid at times shows marked evidence of a former osteomyelitic infection and often shows venous pits. The veins I have been describing are too large to be

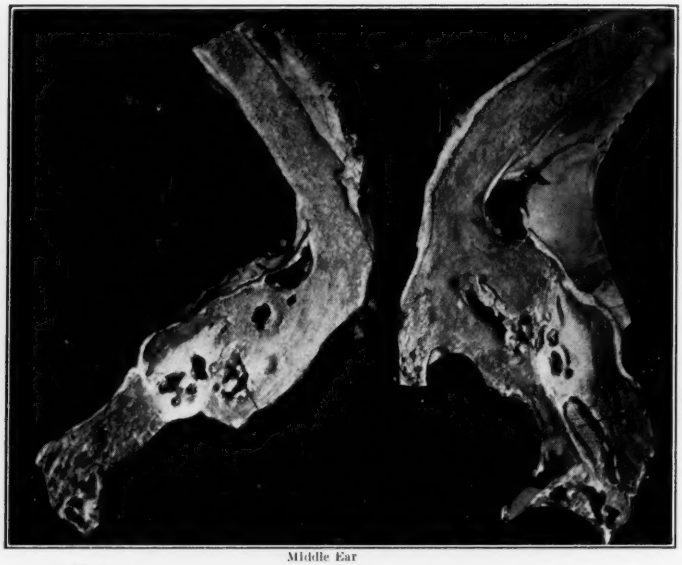


Fig. 9. The illustration shows nicely the apparent suspension of the labyrinth in the temporal bone and the surrounding cells. The marrow spaces and the pneumatic cells of the tip cannot be separated. There is a cellular channel above and below the labyrinth The horizontal portion of the internal carotid was exposed in the making of the section. There are large marrow spaces where the petrous tip joins the sphenoid. The illustration shows how the tip can be reached by following the internal carotid as in the operation of Remadier.

nutrient veins for the bone cortex. They probably are veins from the marrow cells. Many of these openings are pin point in size, others measure 1-2 mm. They run into the bone marrow of the body of the sphenoid or pierce the walls of the sphenoidal sinus into the sinus itself.

CLINICAL APPLICATIONS

The next subject in order is the clinical application of the applied anatomy which I have just given. I shall introduce this by quoting statistics from an article by Courville and Rosenvold on the frequency of intracranial lesions due to nasal infection. This appeared in June, 1938.

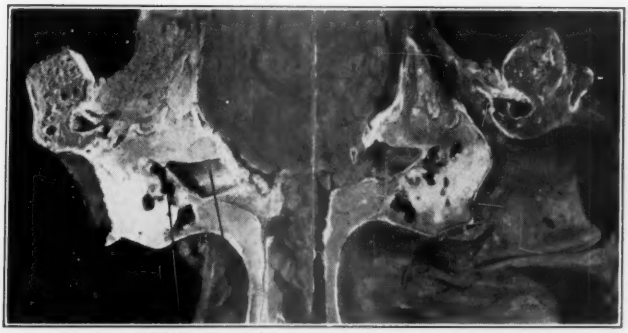


Fig. 10. The illustration shows the apparent suspension of the capsule of the inner ear—seen as dead white in the illustration—in the cellular elements of the temporal bone.

THE FREQUENCY OF INTRACRANIAL LESIONS DUE TO NASAL INFECTION

In the Archives of Otolaryngology, June, 1938, Courville and Rosenvold tabulated 15,000 autopsies, checking infectious intracranial lesions due to diseases of the nasal air passages and the accessory sinuses. The lesions causing meningitis were extra- and intradural abscess—septic thrombosis of the cavernous or the superior longitudinal sinus, pachy-meningitis externa and interna and brain abscess. 62 cases were found. In 11 cases, sinusitis was associated with otitis media. In 52 cases the meningitis was due to an infectious nasal lesion. This is five times the number associated with otitis media.

The frontal sinus was responsible for the intracranial lesion in 14 cases, extradural abscess in four cases, subdural abscess in six cases, and cerebral abscess in only two cases, septic thrombosis of the superior longitudinal sinus secondary to frontal sinusitis occurred in two cases.

There were 18 cases of ethmoiditis, and 22 cases of sphenoiditis. Ethmoiditis and sphenoiditis were frequently associated lesions and their most common intracranial complication was septic meningitis. In the 18 cases of ethmoiditis, 16 were complicated by meningitis, and in the 22 cases of sphenoiditis, 20 were complicated with meningitis.

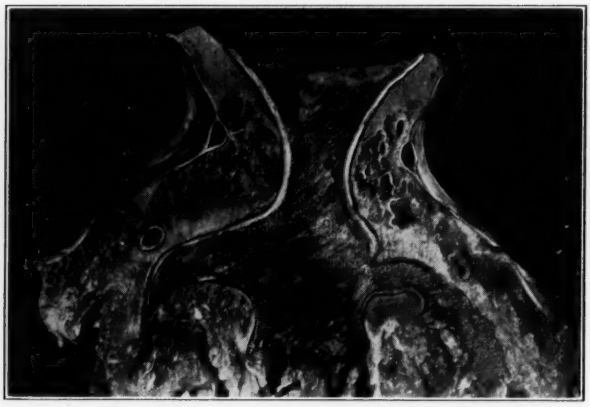


Fig. 11. The illustration shows how the pneumatic cells of the temporal bone and the marrow spaces of the tip resemble each other macroscopically. The marrow spaces extend almost to the internal auditory meatus.

The cases of extra (4) and subdural abscess (6), the two cases of brain abscess and the two cases of septic thrombosis of the superior longitudinal sinus in all probability I feel were caused by infection of the frontal sinus. They total 14, the same as the intracranial lesions said to be due to the frontal sinus alone. The score therefore, stands 14 to 14, showing how common these complications of frontal sinusitis as well as meningitis are.

In this series infections of the frontal sinus as a cause of meningitis has a slight lead, 28 to 20, over the sphenoid.

I have quoted these statistics on meningitis because they show the frequency of meningitis due to nasal infection in a large series of general autopsies. The number seems to us very small, but we are in a special hospital where of course the proportion is many times larger (Fig. 16).

OSTEOMYELITIS OF THE DORSUM SELLAE

Many years ago I had a patient who showed a marked median ridge on the hard palate. The x-ray of the head showed extensive deformity of the dorsum sellae. Had it not been for the exostosis of the hard palate, I should have been worried about the deformity of the sella tursica. Both deformities were assigned at the time to

arthritis. Osteomyelitic or arthritic deformity of the dorsum sellae is so commonly found that no importance is attached to it. Nevertheless, it must have a cause. I am attributing the deformity of the sella tursica to a low grade osteomyelitis from infection originating in the sphenoidal sinus. Sometimes only the crest of the sella tursica is involved, but now and then not only the superior surface of the body of the sphenoid but its inferior surface is involved as well. This to my mind takes the condition and its cause out of the trivial class. Not only is the dorsum sellae often deformed externally but at times the marrow spaces in it are so large that the greater part is hollow. In a number of instances I have found large venous pits opening into it and in one or two cases the pits have communicated with the sphenoidal sinus (Fig. 17).

It is common also to have bony defects in the bases of the internal pterygoid process. They measure in many instances 3 or 4 mm and suggests that an extensive ostemyelitic process is responsible for them.

(See Fig. 19.)

Small knob-like exostoses are common on the cranial surface of the skull. They are most frequent in the anterior half. They attain a large size on the posterior part of the vertical plate of the frontal bone often showing as large transverse bony welts, one above the other. Now and then they are found around the internal auditory meatus and I have often wondered why, in this location, they do not cause trouble (Fig. 18).

The extostoses on the frontal bone have usually been regarded as syphilitic in origin. Undoubtedly some of them are, but I am assigning some or most of them to osteomyelitis caused by the spread of infection from the frontal sinus. We are familiar with the fact the veins of the mucous membrane of the frontal sinus are continued by the diploetic veins of the frontal bone. I have specimens to show that the veins of the mucous membrane of the sphenoidal sinus connect with the marrow spaces of the body of the sphenoidal bone. I have, however, as yet not been able to recognize diploetic veins in the marrow spaces of the sphenoid. If they are absent it may explain the low grade type of osteomyelitis of the dorsum sellae and its chronicity. The term "low grade infection" does not appeal to Dr. MacMillan; neither does it much appeal to me. It is often the resort of the debater who is hard pressed to substantiate his argument. Nevertheless, it can occur and I believe it occurs about the sphenoid. It is striking that the two most frequent places on the inside of the skull for osteomyelitis to occur are the

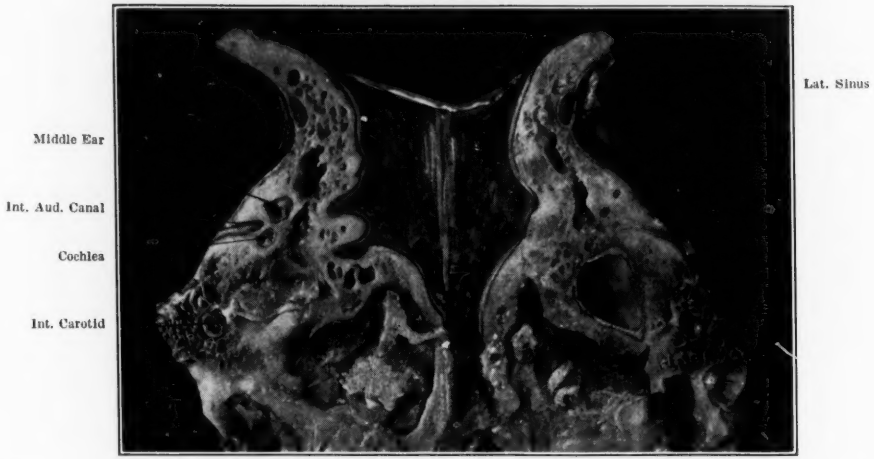


Fig. 12. The cut shows a horizontal section of the temporal bone at the level of the internal auditory meatus. There are many large marrow spaces around the internal carotid artery. Their relation to the sphenoid sinus is shown on the right (reader's right). In this specimen there is a clear anatomical pathway from the sphenoid to the marrow cells of the basilar process and those of the petrous tip.

posterior face of the frontal bone and the posterior face of the sphenoid bone. It is peculair to both that they have nearby mucous membrane from which they can be infected (Fig. 19).

I need this low grade infection of the body of the sphenoid rather than the fulminating infection with which we are more familiar and which is so fatal to bolster up my theory that infection is more often present in the sphenoidal sinus than we suspect and that it can, by low grade bone involvement and by means of the veins piercing its walls, transfer infection to the gasserian ganglion and thus give a neuritis of the ganglion and cause retro orbital pain.

HOW DOES INFECTION FROM THE SPHENOIDAL SINUS REACH THE BODY OF THE SPHENOID OR THE PETROUS TIP?

I have just given my answer to this question. It was that the veins of the mucous membrane of the sphenoidal sinus are connected with the marrow spaces of the body of the sphenoid. Infection can travel by the veins into the body of the sphenoid or the sella tursica or dorsum sellae.

There is a characteristically splendid article in the Archives of Otolaryngology, Vol. 30, No. 2, by Blast and Forester on "The Origin and Distribution of Air Cells in the Temporal Bone." I quote from the summary on page 205 the following:

"Large spaces in bone marrow resembling air cells are sometimes seen in histological sections, but they are not true air cells. They are due either to some pathologic change or to postmortem formation of gas."

My comments on these statements are as follows:—To the naked eye large spaces in the bone marrow (of the petrous tip) which have a provoking resemblance to air cells are not "sometimes seen", but often seen. I am glad to find that the writers feel that these large spaces in the marrow may be due to "some pathological change." This is my belief, especially as regards the large spaces found in the marrow of the tip of the temporal bone and in the basilar process of the sphenoid.

In the specimen shown in Figs. 17-18 there is marked evidence of old infection as shown by the osteomyelitis of the upper part of the basilar process of the sphenoid and of the dorsum sellae, and in addition, by the large perforations through the cranial surface of the basilar process. The perforations communicate with unusually large marrow spaces in the basilar process of the sphenoid and one with the sinus itself. I believe that the infection respon-

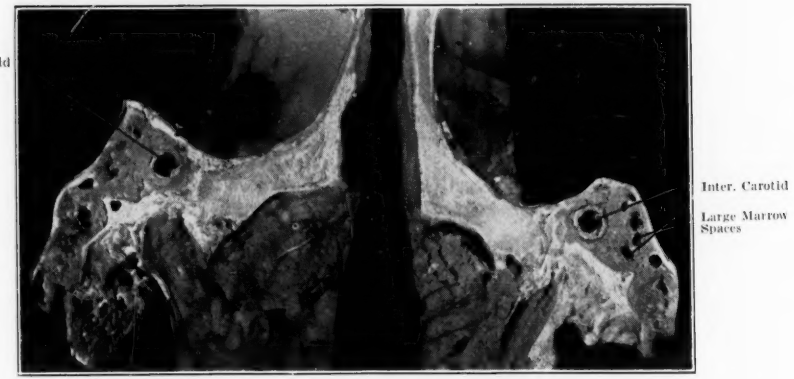


Fig. 13. The illustration shows large marrow spaces in the basilar process of the sphenoid. They are near the internal carotid, and directly under the cranial surface of the basillar process. The neighborhood of the internal carotid is a favorite place for large marrow spaces to group. (See Fig. 12.)

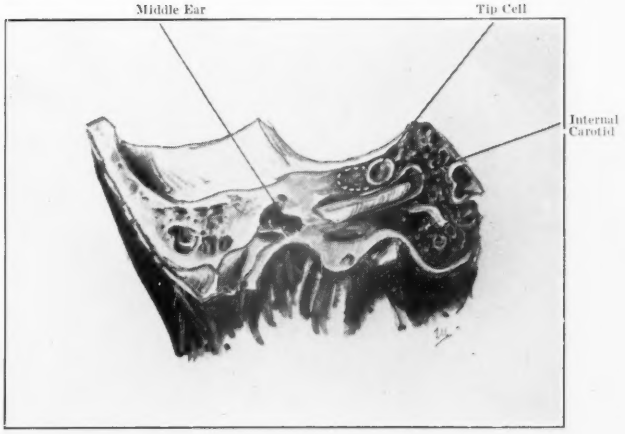


Fig. 14. Natural size. The cut shows a horizontal section through the temporal bone at the level of the internal auditory meatus. The horizontal portion of the internal carotid artery was exposed by the cut. Above the artery there is a large tip cell. Its size is indicated by the dotted white line. There are many good sized marrow spaces about the inner end of the artery and beyond it.

sible for these changes originated in the sphenoidal sinus, and was carried from the mucous membrane of the sinus to the marrow spaces, and is similar to what happens in the frontal bone. I believe further that infection of the petrous tip can originate in the same manner (Figs. 20-21).

RETROBULBAR PAIN

There are cases of retrobulbar pain not associated with otitis media or infection of the petrous tip. These I feel are due to infection in or about the Gasserian ganglion without demonstrable pus and with a clear petrous tip to x-ray.

These cases so far have remained unexplained. The frequent osteomyelitic changes in the dorsum sellae and the body of the sphenoid bone also remain unexplained.

Exostoses of the posterior surface of the perpendicular plate of the frontal bone are common, and more common in this locality than in other parts of the skull. Infection from the frontal sinus can be

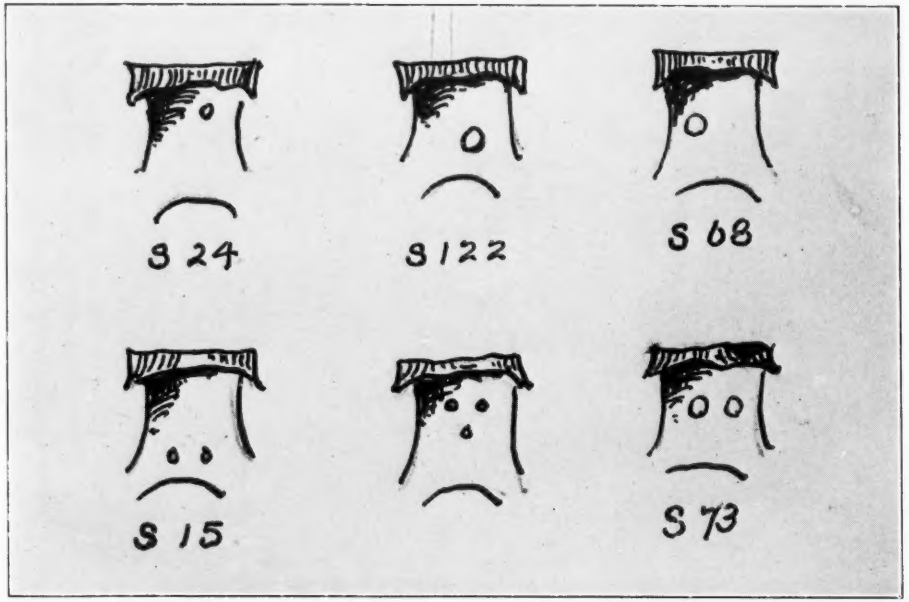


Fig. 15. The illustration shows the dorsum sellae and the basilar process of the sphenoid from six dried skulls. They all show one or more perforations in the basilar process. The illustrations are roughly natural size and give the number, size and position of the perforations of the cranial surface of the basilar process.

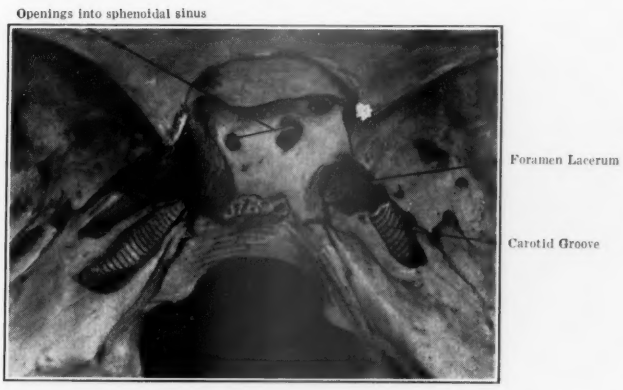


Fig. 16. The illustration shows two large openings through the sella tursica into the sphenoidal sinus. Openings of this size the writer feels indicate old infection.

assigned as the probable cause. Infection from the sphenoidal sinus can in like manner infect the marrow spaces.

Often the infection about the gasserian ganglion is not virulent enough to cause actual bone destruction or meningitis. It clears up after a period and the pain disappears to reappear if the infection lights up again. There can be an infectious neuritis without pus. This is common in other parts of the body and causes no surprise. A chronic osteomyelitis of the petrous tip—and there is such a thing as Dr. MacMillan and others have shown—could also be the infecting focus for the ganglion as well as infection from the ethmoid or sphenoid.

For some time now we have accepted without question gasserian ganglion neuritis as a natural complication of infection of the petrous tip. Right or wrong, I have advanced a possible, yes a plausible explanation for gasserian ganglion neuritis without infection of the middle ear or mastoid. Eagleton has been right many times and never more right than when he said watch the sphenoid.

How far have we reached tonight? We started off with a review of some of the fundamental points in the anatomy of the temporal bone stressing especially the cellular pathways, or rather the lack of them, to the petrous tip except in extensively pneumatic bones, such being the findings in 83 dissecting room heads.

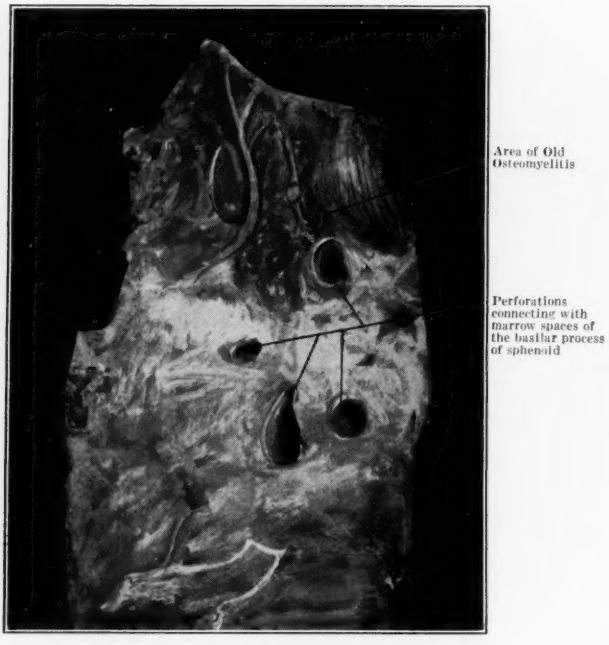


Fig. 17. The cut shows the cranial surface of the basilar process of the sphenoid. In the upper part of the specimen there is an area of old osteomyelitis. Below this there are four perforations, each one leading to a large marrow space in the basilar process. (See Fig. 15.)

Then the apparent extension without break of the marrow spaces or cells of the body of the sphenoid into the tip of the temporal bone was discribed, such cells or spaces often extending to the internal auditory meatus of the temporal bone. This led to the natural question, why infection of the marrow cells from the sphenoidal sinus could not be a cause of petrositis. Eagleton's contention that petrositis was often an osteomyelitis of the marrow cells rather than an osteitis of pneumatic cells was held to be reasonable.

It was suggested that deformity of the dorsum sellae which is so common as to be usually considered as of no significance is a sign of old osteomyelitis of the body of the sphenoid, the infection back

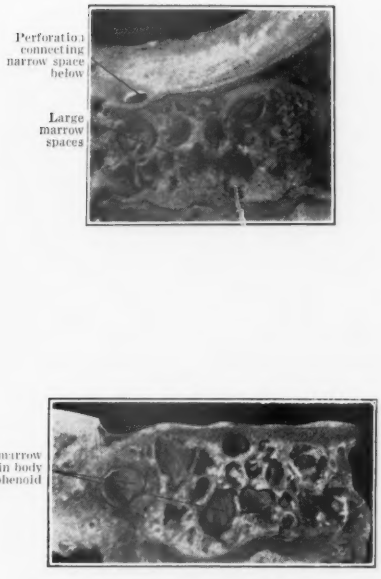


Fig. 18. Both cuts are from a cross section of the basilar process of the sphenoid. The marrow spaces are very large. The upper cut shows a perforation in the cranial surface of the basilar process which leads to a marrow space below. There are four such perforations, and each connects with a marrow space. (See Fig. 15.)

of it originating in the mucous membrane of the sphenoidal sinus. If this is the true explanation then we have in repeated non-fulminating infections of the sphenoidal sinus the cause of infectious neuritis of the gasserian ganglion and so a cause of retrobulbar pain without infection of the petrous tip.

Non-fulminating infection of the sphenoidal sinus—I like this phraseology better than the term "low grade infection"—might or might not show in the x-ray. The treatment would not be necessarily radical surgery. A trial would first be made to free the front face of the sphenoidal sinus sufficiently, by minor surgery on the posterior ethmoidal cells in cases where this proved necessary, so that intrasinus medication by the Proetz method could be employed.

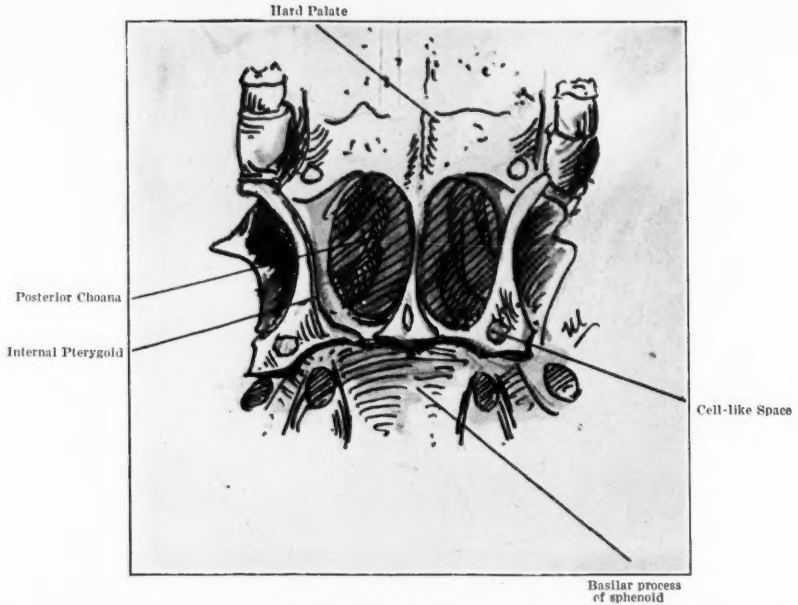


Fig. 19. The specimen shown in the illustration has a large cellular space at the base of each internal pterygoid process. These spaces are rather common. They suggest old bone pathology, especially where they are multiple and confined to one side, as they often are. In the specimen here illustrated they are probably an anatomical variation. These spaces at the base of the internal pterygoid process are new to the writer and are so striking that they are mentioned in passing, so to speak. They will bear watching.

PART II

In my early days in otolaryngology petrositis was regarded as a rather rare complication of mastoiditis. Gradinego's syndrome was known, of course, and considered in great part only a dramatic happening. The cause of the syndrome was rightly guessed, but the full meaning of the pathological conditions back of the syndrome was not sensed, neither were the possible complications acutely realized. This haze was due to the fact that in some cases the symptoms cleared up of themselves or subsided after a simple mastoid operation.

Now and then a case would die of meningitis. Even this happening did not lead to a systematic effort to clear up the fog surrounding these cases. I, for one, got no further toward an explanation than to feel that there must be some method of drainage from the petrous tip to the middle ear and the mastoid cells, probably by lymphatic channels, which allowed spontaneous healing in some cases, in others was helped and made effective by a thorough mastoid operation. What has happened since this far off time is too well known to be more than touched upon here. The reader knows that it has been fully proved that there is drainage by cellular channels from the petrous tip to the middle ear and to the mastoid itself, resulting at times in the life saving formation of a fistula. Literally the whole world is now fully alive to the possibility of meningitis as a fatal complication of petrositis. The petrous tip has been attacked surgically from all available sides. To speak figuratively there is a sniper blazing away at it from every anatomical window and roof top. It will soon be seen that I am sniping myself. As yet there is no simple, easy approach, with perhaps the exception of the fistula route—to the petrous tip. No approach yet devised is fully an approach by sight.

Another observation which has come out of late years is that there are cases of chronic petrositis. Dr. MacMillan has had a chance to watch a few such cases over a period of years and finally has seen the infection resolve. These few cases were mostly patients who refused operation or were seen in the early days before the gold rush of petrous operating began.

The following is the summary of the case which these introductory and slightly reminiscent remarks have been leading up to. The discussion of this case forms the second part of the paper.

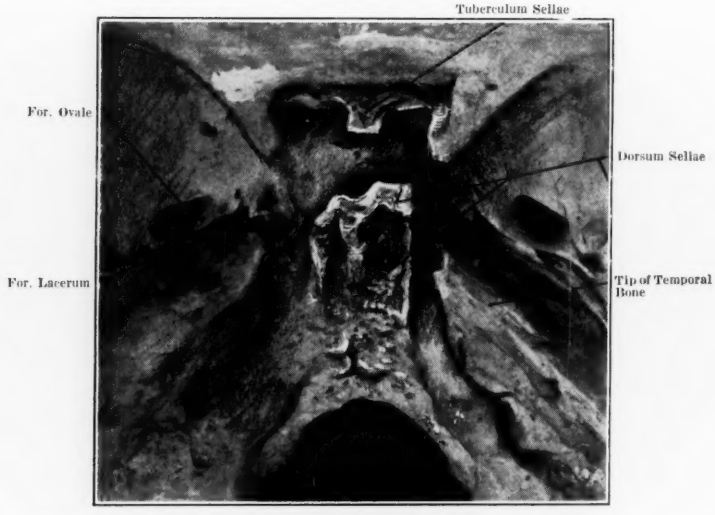


Fig. 20. The illustration shows healed osteomyelitis of the dorsum sellae. The tuberculum sellae also shows slight involvement. Such changes have usually been called arthritic, and as they are rather common, they have been dismissed as of no importance. I have taken an opposite view in this paper. I consider the changes due to old infection originating in the mucous membrane of sphenoidal sinus.

SUMMARY OF HISTORY

Frank Mulvey—34 years.

November 4, 1938: Ten weeks before entrance patient had acute otitis media. Pain, two weeks. Discharge, two days. Tinnitus, ten weeks. Deafness, ten weeks. Headache, six weeks left side and chiefly occipital. X-rays show destruction in mastoid cells.

November 5: Operation. Dr. Fred and Dr. Drooker. Simple mastoid operation with exposure of lateral sinus and dura. Both were macroscopically normal.

November 9: Recurrent profuse discharge.

November 28: Twenty four days after admission and about three months after the original acute otitis media, the x-ray of the right mastoid was normal. The left mastoid showed a post operative defect. The petrous tip was decalcified.

November 29: Operation. Dr. Fred. Revision of left simple mastoid. Normal granulations over dura and lateral sinus. Normal bone found in solid angle. No fistula present.

December 7: X-ray report—no further involvement of petrous tip.

December 13: Postaural wound clean.

December 15: Closure of postaural wound.

December 19: Patient was discharged to Out-Patient Department.

December 29: Readmitted to the House.

Interval History: After absence of ten days patient returns to the hospital because of swelling of the left side of the neck, together with stiff neck. These symptoms began five days ago. Had been without symptoms up to five days ago. Has had no fever, chills, nausea, vomiting nor vertigo since dismissal.

Local Examination: Eyes normal. No external rectus paralysis. Slight left torticollis. No actual neck swelling. Nose normal. Throat: left tonsil pushed somewhat toward the middle line. Left pharyngeal area appears full. Left ear: postaural incision open at lower end. No pus. Membrana tympani is dull. Right ear: Membrana tympani yellow. Secretion in the middle ear.

Summary: Left ear—Pain five days since discharge from hospital. Discharge since October 20, 1938. Tinnitus, sixteen weeks. Deafness, sixteen weeks. Headache, chiefly occipital 12-14 weeks. Nausea, none. Sweats, none. Vomiting, none. Chills, none. Fever, none. Postaural tenderness, two days. Postaural swelling, five days.

December 30: Lumbar puncture; initial pressure 135, clear—no cells. Spinal fluid culture negative. X-ray report—There is more decalcification of petrous apex. There is prevertebral swelling from the base of the skull to the level of the thyroid cartilage.

December 31: Tenderness at angle of jaw on left. There is fullness above the left tonsil and the tonsil is pushed toward the middle line. Incision through left supratonsillar fossa gave no pus.

January 2, 1939: Eye consultation. Discs and fields normal.

January 4: Patient still had headache. Complains today of nausea for the first time. Spinal fluid Wassermann negative. White blood count 15,000; polymorphonuclears 72%; lymphocytes 22%.

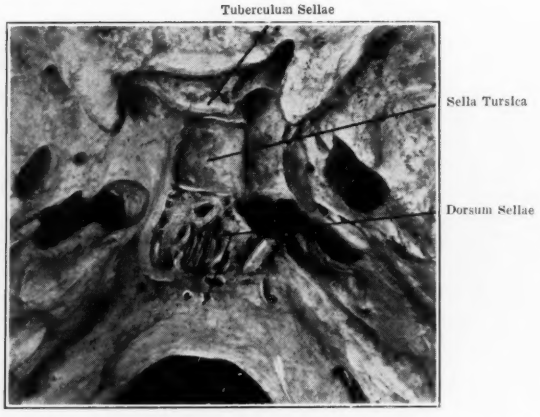


Fig. 21. The illustration is from a specimen-dried skull in which there has been marked osteomyelitis of the dorsum sellae. The tuberculum sella also shows the same condition, but to a lesser extent. (See Fig. 20.)

Operation, Dr. Mosher and Dr. Mysel. Exploration of left pharyngo-maxillary fossa.

January 12: Wound healed except at lower end of the drainage site.

January 14: X-ray report: Less swelling in nasopharynx. Petrous remains the same.

January 25: Neurological examination negative.

January 31: No headache or other symptoms.
February 1: Discharged.

Out-Patient Department.

March 15: No symptoms. Left drum normal (Fig. 20).

September 6, 1939: Mulvey reported at the hospital today by request. It is now thirteen months since his first ear involvement and eight months since his last operation. He has been entirely free from all symptoms since the last operation. The x-ray of the petrous tip taken today shows no sequestrum. The left petrous tip is still hazy but is becoming calcified again. There are no signs of infection.

Throughout this case the chief symptom was headache which was mostly occipital. The cessation of the postaural discharge after the mastoid operation with its return later in volume suggested the petrous apex as the reservoir from which it came since the x-ray showed petrositis. Apart from a non-typical headache the cardinal symptoms of petrositis were not present, especially paralysis of the sixth nerve. There was no fever and no signs of meningeal infection.

In this patient the petrositis was of the mulling, almost chronic type. It has been found that such cases can at times clear up of themselves. The finding in this case which showed that the process was not resolving but slowly progressing, was the swelling of the side of the neck, the lateral pharyngeal swelling with the pushing of the tonsil toward the middle line, and the x-ray finding of swelling in the prevertebral space from the base of the skull to the thyroid cartilage.

After a mastoid operation the appearance of headache suggested beginning meningitis or brain abscess. In the old days when a mastoid had been exenterated and the discharge had abated or ceased, only to start again in volume, this was regarded as one of the signs of a brain abscess. The amount of the pus was too great to come from the middle ear or the exenterated mastoid cavity and the most likely place for it to come from was the cranial cavity, either superficial or deep to the dura, or from the substance of the brain itself. Today we recognize that the renewed discharge can come from a reservoir in the petrous tip and probably more often comes from here than from inside the cranium.

It was the x-ray that gave the diagnosis of infection of the petrous tip and substantiated the clinical finding that the infection was making its escape into the vault of the pharynx. For many years now, post-pharyngeal swelling has given a measure of the post-pharyngeal infection after the swallowing or the extraction of foreign bodies lodged in the pharynx or in the esophagus. In the case under discussion the post-pharyngeal swelling located the source of the infection and charted its course. It was a great satisfaction to watch it gradually subside, after drainage had been established. The lateral x-ray not only showed the post-pharyngeal swelling but disclosed that the dorsum sella was markedly enlarged and deformed, and there were perforations in the basilar process of the sphenoid. This fits in with my theory, that there had been old trouble in the sphenoid sinus and raises the question as to what part the lighting up of this infection played in the present condition. Occipital headache

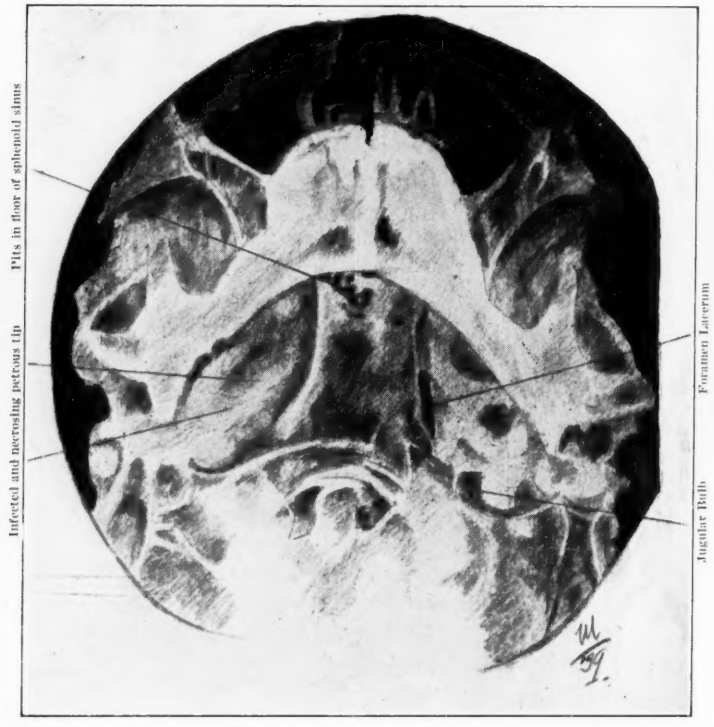


Fig. 22. Mulvey. Tracing of an x-ray film. On the left it shows marked fogging of the petrous tip. Everything in the basal fossa of the tip is blurred, especially the foramen lacerum. On the right the edges of the bony markings are clear cut. The foramen lacerum stands out distinctly. At the upper part of the basilar process there appear to be a number of erosions on the pharyngeal surface. A repeat x-ray which showed more of the sphenoidal sinus, in fact the greater part of it, made it clear that these apparent erosions were irregularities or pits in the bottom of the sinus. At first the writer considered them osteomyelitic erosions. This was an example of wishful thinking which was fortunately corrected in time. (See Fig. 24.)

which was the type which the patient had is characteristic of sphenoid disease. This again points to the sphenoid as playing a part in the etiology of this case (Fig. 22).

From my experience with infection in the pharyngomaxillary fossa I have long been wedded to the submaxillary approach to pus high in the neck, especially as this approach allows the operator to reach the base of the skull easily, if it is necessary to go so far in the search for the pus. Naturally, therefore, I turned to this approach

in order to determine whether the post-pharyngeal swelling was inflammatory only or due to the actual presence of pus. I was anxious also to find out whether or not the tip of the petrous when approached in this manner, could be palpated and recognized. To my great delight this proved to be the case. It was bare and rough (Fig. 23).

The exploration over, I waited to see what, if anything, had been accomplished beyond learning the condition of the under surface of the petrous tip and the presence or absence of pus at this point. To make a long story short the patient's headache disappeared in two or three days and has not returned to date. The post-pharyngeal swelling disappeared in a week or ten days. Naturally we discussed what the next step would be should symptoms return or the x-ray show the formation of a sequestrum. In either eventuality I favored operation by the middle fossa route. Unless a fistula can be found draining the petrous tip into the middle ear and so guide the operator to the tip, I am more and more in favor of the attack on the petrous tip by sight, in other words, by the middle fossa route. In the exceptional case, like the one here reported, where the tip is draining into the pharynx, I feel that the submaxillary approach to the base of the skull should be given further trial.

After this dramatic case I got to the Medical School and my specimens at the first opportunity, that is, on the next day, in order to refresh myself on the anatomical landmarks at the base of the skull in the region of the petrous tip. I found as I suspected, that the finger skirting the inside of the styloid process and carried to the base of the skull naturally touched the under surface of the petrous tip. There is a fossa at this point which neatly takes the end of the finger. This fossa is bounded on the outside by the styloid process, on the inside by the tip of the foramen magnum and anteriorly by the under face of the carotid canal. On this, there is often a sharp ridge which runs obliquely from behind forward to the foramen lacerum. This line or ridge when present is readily felt and makes the anterior boundary of the fossa into which the exploring finger falls. It is into this fossa that pus escaping from the lower surface of the petrous tip evacuates and gains the pharynx (Fig. 24).

At this point some one will ask, suppose you find pus at this point, what can you do or rather what would you dare attempt, knowing the nearness of the internal carotid artery? Would you dare to curette? I should be loath to do so. Dr. Wherry, Senior, suggested the natural expedient of carrying a lighted speculum or

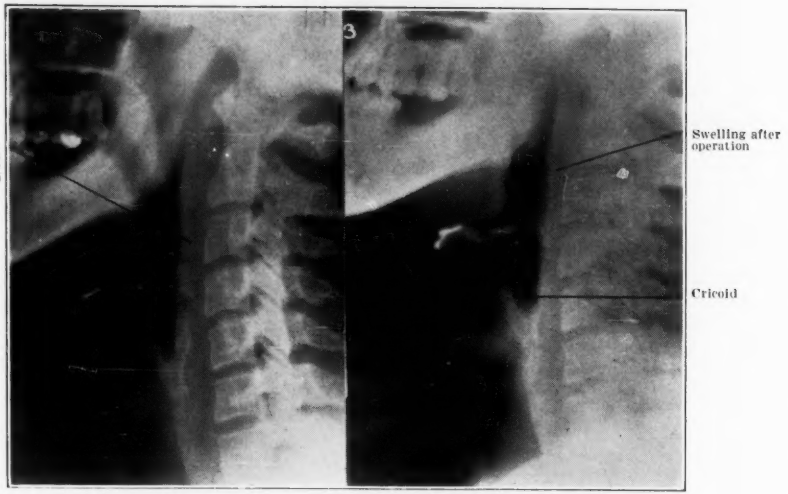


Fig. 23. Mulvey. X-ray before and after operation to show the postpharyngeal swelling. This rapidly decreased after exploration of the base of the skull by the pharyngomaxillary route.

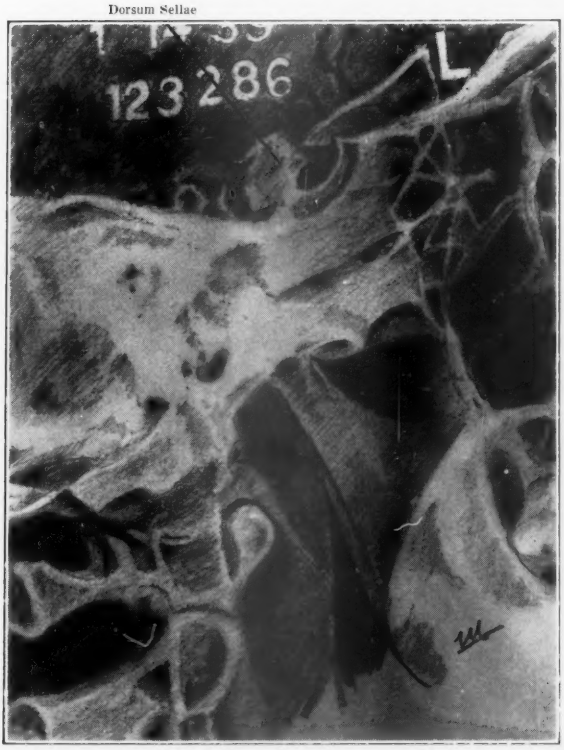


Fig. 24. Mulvey. The illustration is a retouched print from an x-ray. There is marked deformity of the dorsum sellae. The writer holds that this is evidence of an old osteomyelitis. This was found in a re-examination of the x-ray films long after the patient was discharged from the hospital the last time. (See Fig. 22.)

a lighted tube of appropriate size to this point and get what help this might give. As yet I have had no experience with this on the living, but on the cadaver light can be carried to the point in question with the head mirror and speculum or with a lighted tube. Should the opportunity present itself again, I shall certainly try this expedient.

Eagleton—to whom I am trying desperately throughout this paper to give full credit—advocated in cases where there is a retropharyngeal evacuation of pus from the petrous tip, to incise the

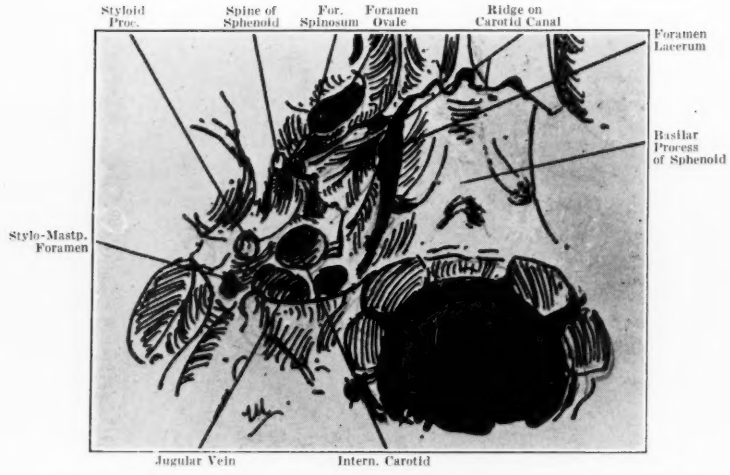


Fig. 25. The illustration shows the anatomical landmarks surrounding the under surface of the tip of the petrous pyramid.

abscess swelling through the roof of the pharynx. This connects the petrous tip with the pharynx, a filthy place at best. Myerson has a cross country route to the under surface of the petrous tip from the side of the neck high up. I am not taking violent issue with either gentlemen, but to me the submaxillary fossa route through the pharyngomaxillary fossa though it may seem a bit long to one who tries it for the first time, avoids opening through the dirty pharynx, is direct, and has the possibility of operating under direct vision. It is following nature's route as the x-ray shows and nature is no mean guide to follow (Figs. 23-24).

The anatomy of the petrous tip is complicated. My suggestion in this matter, to bring the discussion to a close, is that the reader take the diagram shown in illustration number 25 and study it in connection with a cleaned skull. Then follow this by trying the submaxillary approach thorugh the pharyngomaxillary fossa to the petrous tip on a wet specimen and be prepared for the next case where pus from the petrous tip makes its way into the upper pharynx. I am strongly of the opinion that the success of the procedure in this case means that it is something more than a fortunate happening and that it should be given further trial by competent hands.

FINAL SUMMARY

The first part of the paper—the talk given to the House Staff has already been summarized. In the light of the case just reported, which forms the second part of the paper, is there anything to add? Simply this,—The case proves that the pharyngomaxillary approach to the petrous tip is feasible, direct and more under the control of vision. Further, it shows that there had been old infection of the sella tursica. The case has made the petrositis picture more complete, at least to me. As I see the picture now a final summary would run as follows:—The tip of the petrous pyramid can be infected from the mastoid cells by the cellular routes around the labyrinth which are pretty sure to exist in a markedly pneumatic temporal bone. The tip can be infected by the venous route, namely by way of the lateral and inferior petrosal sinuses. It should be remembered that the inferior petrosal underlies the whole length of the tip. The tip can be infected by infection spreading from the sphenoidal sinus into the marrow spaces of the body of the sphenoid because these spaces are so often continuous with the marrow spaces of the petrous tip. The infection, whatever its source, may result in an osteitis of the pneumatic cells of the tip or an osteomyelitis of the marrow spaces. In an advanced case both are probably involved together. Depending on its virulence the infection disregards histological barriers. In advanced cases the pneumatic cells and the adjoining marrow spaces break down together and form a surprisingly large reservoir for pus. In time this finds its way into the middle ear with or without a fistula demonstrable at operation and accounts for the recurrence of an abundant discharge. Finally an acute or chronic focus of infection in the sphenoidal sinus can produce an infectious neuritis of the gasserian ganglion with intermittent or continued retro-orbital pain. This is in every way similar to infectious neuritis common in other parts of the body. This explains retro-orbital pain and with or without paralysis of the Sixth Nerve and without ear infection. Throughout this paper the sphenoid has been featured as a source of infection for the petrous tip and the gasserian ganglion. It should be remembered, however, that the ethmoid and the vault of the pharynx also must be reckoned with.

127 FRONT STREET,
MARBLEHEAD, MASS.

II

METHODS FOR PRODUCING LOCAL ANESTHESIA FOR TONSILLECTOMY, INTRANASAL OPERATIONS AND OPERATIONS ON THE PARANASAL SINUSES*

HAROLD I. LILLIE, M.D.*

PETER N. PASTORE, M.D.*

AND

LLOYD H. MOUSEL, M.D.†

ROCHESTER, MINNESOTA

Local anesthesia for tonsillectomy,[4] intranasal operations and operations on the paranasal sinuses has been used successfully for a long time. Much has been learned from the reported experience of observers who have described methods of safely producing anesthesia by means of cocaine and its derivatives. That there are many features involved in the successful use of local anesthesia in any field of surgery must at once be obvious. No claim for originality in method is made, because it is realized that the technics employed are combinations of several methods and are used as such because the results have been increasingly satisfactory as experience has increased.

Selection of Anesthesia: The choice of anesthesia for tonsillectomy or intranasal operations should not be left entirely to the patient's preference, because the surgeon knows whether or not an operation can be performed more satisfactorily under local or general anesthesia and it should, therefore, be his prerogative to designate the anesthetic to be used. This need not be done in an arbitrary manner. The greatest objection patients have to local anesthesia is that they are afraid they will feel too much pain if it is used or that they do not wish to realize what is taking place during the operation and prefer to be asleep. At The Mayo Clinic local anesthesia is used for tonsillectomies and intranasal operations on many adolescent patients and, with few exceptions, all adult patients, because local anesthesia has been found eminently satisfactory by the individual surgeons for their individualized technic.

---

*Section on Otolaryngology and Rhinology, The Mayo Clinic.

†Section on Anesthesia, The Mayo Clinic.

Contraindications to operation or anesthetics: Whenever it is felt that considerable improvement in the patient's general condition may result from removal of infected tonsils and treatment of diseased sinuses, there seem to be few contraindications to surgical treatment so far as the general condition is concerned. This is said because many imperfectly informed persons insist on tonsillectomy and surgical treatment of sinuses in the presence of conditions in which the surgical risk is too great and the chance of improvement is too small. Hemophilia, malignant hypertension and seriously advanced general diseases are obviously definite contraindications to surgical operation.

Obviously, certain patients may have an idiosyncrasy to cocaine and its derivatives. This fact can be elicited by questioning or by trial. Should the patient have had any terrifying experience previously as the result of use of cocaine, he will be sure to mention the fact. Therefore, it is necessary to determine by test if an idiosyncrasy actually exists. An overdose of epinephrine may have been the causative factor. However, enough genuine idiosyncrasies are encountered to warrant investigation of the patient's sensitivity before local anesthetics are used. To test for sensitivity, the forearm should be carefully cleansed with alcohol and allowed to dry. A small intradermal wheal should be raised, using a physiologic saline solution to raise a control wheal. A series of intradermal wheals should then be raised on the forearm with the various drugs that are to be used. The degree of dermal sensitivity present may be determined by observing the extent of reaction around the wheal as compared to that of the control wheal. If a reaction to any injected material occurs all thoughts of use of that particular drug should be discarded and another drug substituted.

In case of occurrence of syncope, due to emotional reaction and not to the effect of drugs, the patient's head should be lowered before any local anesthetic is used. Cold compresses should be applied to the forehead and the back of the neck. Inhalations of aromatic spirits of ammonia seem to be of benefit. Syncope usually lasts but a few minutes.

The acute symptoms of sensitivity to local anesthetics vary with the drug used, but in general there are certain toxic manifestations that are evident in varying degrees of effect. Toxic effect on the vasomotor system is manifested by pallor, faintness, perspiration, rapid weak pulse, irregular cardiac action, lowered blood pressure, headache, and vomiting. Respiratory manifestations may be sudden hunger for air and acute pulmonary edema. Effects on the nervous

system are exhibited by excitement, hilarity, garrulousness, incessant talking, crying, apprehension, spasms or tremors, convulsions and paresthesias.

Death from cocaine poisoning may occur suddenly.[7] One of us (Lillie) as a house officer observed a delayed reaction to cocaine that resembled scarlet fever. Although this type of reaction is rare, the physician must always be on the alert, for the progress of the train of symptoms from a mild beginning to convulsions is often rapid. A sterile packet containing pentobarbital sodium, triple-distilled water, syringes and needles, should be kept on hand for just such an emergency. Should a reaction occur, a solution of 2.5 per cent pentobarbital sodium should be injected intravenously at the rate of 1 to 2 grains (0.065 to 0.12 gm.) per minute until the patient has been relieved of his convulsions and a light degree of anesthesia has been produced.

Knoefel, Herwick and Loevenhart collected from the literature 593 cases in which the administration of barbituric acid preceded that of local anesthetics. Four of the cases were reported as indicative of mild intoxication. Those cases in which the drug was not successful are presumably those in which absorption of the drug was rapid. The authors stated "that the susceptibility to cocaine and other local anesthetics, on subcutaneous administration, varies directly with the brain development; that is, the ratio of brain weight to body weight. However, these considerations become reconcilable when we consider that on subcutaneous injection the first organ to respond to the small amounts of the local anesthetic in the blood is that which is the most sensitive, namely, the brain, while the first organ to respond to the large amounts of local anesthetic thrown directly into the blood, as by intravenous injection (experimental animals), is that reached first, namely, the heart."

Some surgeons do not use local anesthesia successfully because they are unfitted to do so. Unfortunately, there are some who do not realize, or will not recognize, their limitations, and as a result condemn the method as used by others. It is necessary and important that the surgeon have confidence in his own ability, so that he may instill a feeling of confidence in the patient, and thus perform the operation successfully. Under these circumstances, the undercurrent of uneasiness or dread is relieved immediately to a large degree and the patient and surgeon stand on common ground. In such a case, the surgeon is calmly at ease, as dignified as the situation may demand, pleasant, cheerful, kind, adaptable, and resourceful, for he must be in control of the whole situation. Uneasiness, fret-

fulness, or excitability do not instill confidence. What could be more tragic than an irritable, incompetent surgeon operating on an excited, resisting, non-coöperating patient?

The patient about to subject himself to an unpleasant experience because of possible benefits to his health accruing therefrom knows or at least, if inquiry has been made, has been advised, that there is always an element of danger in any surgical operation, even under the best conditions. The human being has an intellect. Persons have been known (or have been said) to have died from fright; therefore, it is incumbent on the surgeon to control his patient's emotions. There is great chance for drawing erroneous conclusions concerning the effect of drugs from results of experiments on laboratory animals like the frog, because the reactions and emotions of the human being are much more complex than those of the experimental animal.

Types of Patients Encountered: Patients who undergo operations under local anesthesia can be classed into four main groups as determined by personality and emotions, and those in each group must be dealt with differently. It becomes relatively easy to classify patients to one's own satisfaction before operation, and to be forewarned is to be forearmed.

In the first group, the patients are intelligent, unafraid and confident, and will submit to operation in a spirit of complete coöperation, both mental and physical. Pharyngeal reflexes are easily controlled and suggestions are obeyed exactly, even in spite of any annoyance. Operation is easily performed, even though some technical difficulty may arise. The surgeon, outwardly at least, must be perfectly at ease.

In the second group, the patients are of the same general type as in the first group except that despite their efforts they have difficulty in controlling pharyngeal reflexes. This adds to the technical difficulty of the operation. The patients realize the condition and invariably say that they are sorry and that the surgeon should proceed. The surgeon pays little or no attention to the pharynx and performs the operation as expeditiously as possible. Usually, these patients are most appreciative.

In the third group are the nervous, fearful, perhaps tearful, patients. Try as they will, they have little control over their emotions. Realizing this, they increase the difficulty. They have confidence in their surgeon, but are still afraid. Under these circum-

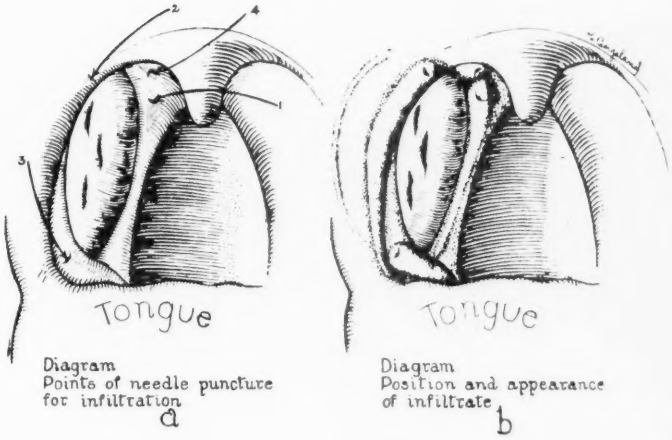
stances, local anesthesia is not complete without "vocal" anesthesia, that is, the surgeon must be kind, buoyant and gentle. To relieve the tension, he may joke with the patient, or at least make an effort to make him smile. A continuous chatter while he works often has a good effect; whistling softly or singing may entirely relieve the situation. What might be called a "scientific indifference" on the part of the surgeon will greatly relieve the difficulties. After operation, the patient's state of mind is entirely changed.

In the fourth group are the purposely noncoöperating, sullen patients. Their attitude may be the result of fear, but this does not seem to be an obvious cause. Under such circumstances, the surgeon must be stern and exacting rather than cheerful and buoyant. Operation under such circumstances is occasionally an ordeal for both patient and surgeon.

Preoperative Care: When the operation indicated has been decided on and the patient has been submitted to a general examination, so that the surgeon may be forewarned of any serious physical condition, the patient is sent to the hospital the evening before operation. This has been found advisable because it places the patient more at ease and allows him to become accustomed to his surroundings. The patient's temperature is checked the evening before and the morning of the operation. All patients more than fifteen years of age receive one and a half grains (0.097 gm.) of pentobarbital sodium at bedtime. This drug will ensure the patient a good night's rest without apprehension. The morning meal is omitted.

The morning preoperative medication or basal anesthesia differs according to the type of operation involved. For tonsillectomy, one and a half grains (0.097 gm.) of pentobarbital sodium is administered one hour before the scheduled time for operation. Atropine sulfate, 1/150 grain (0.0004 gm.) and morphine sulfate, 1/6 grain (0.01 gm.) by hypodermic injection is administered just before the patient leaves his bed for the operating room. This procedure generally allows from fifteen to thirty minutes for the drug to take effect. At the end of this period the patient is prepared for the local anesthetic to follow.

In preparing the patient for submucous resection, intranasal sinusotomy, external operation on the frontal sinus and the Caldwell-Luc operation, the preoperative routine is identical to that outlined for tonsillectomy, with the exception of administration of pentobarbital sodium. For the aforementioned procedures, pentobarbital sodium in doses of one and a half grains (0.097 gm.) is administered



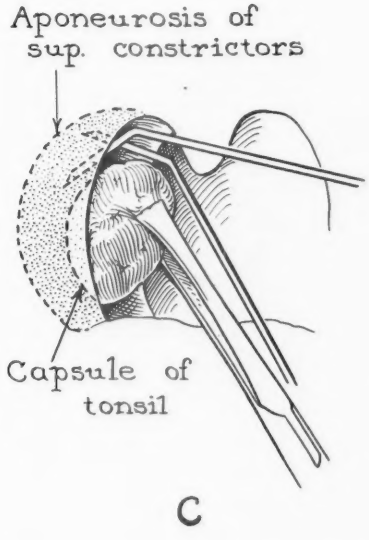


Fig. 1a. Numerals 1, 2, 3 and 4 indicate points of injection for tonsillectomy; *b*, sketched appearance of infiltrate, and *c*, capsule of tonsil being held by forceps.

two hours and one hour, respectively, before operation. Patients who are to undergo external frontal sinusotomy or the Caldwell-Luc operation are administered in addition, one and half grains (0.097 gm.) of pentobarbital sodium, just before being brought into the operating room. In addition, morphine, 1/6 grain (0.01 gm.), may be given any time during the operation if necessary. If the effect of the basal anesthetic is more pronounced than necessary (an occurrence that happens only occasionally), the operation can be delayed until the following morning and smaller amounts of the basal anesthetic can be given at that time.

It has been found best, if possible, to usher the patient into the operating room without the use of a wheel chair or any other conveyance, so as not to break down the patient's sense of personal reliance. There must be an atmosphere of cheerfulness in the operating room. The surgeon, assistant, and nurses should show no signs of anxiety if any is felt. In procedures that do not require the use of an operating table, the patient should be seated in the upright position in a chair suitable for operation and calmly instructed how to coöperate. If pallor, syncope, nausea or petit mal are manifested before the use of any anesthetizing agent, they will be largely due to emotion and the patient's head should be placed between his knees until he feels better. The patient, considerably reassured because nothing has happened, should be instructed to relax completely and to breathe through the mouth. From this point on, the situation usually will be well under control of the surgeon.

Anesthesia for Tonsillectomy: It is not necessary to paint the faucial pillars, pharynx and tongue with an anesthetic solution; it is preferable that the pharyngeal reflex be maintained so that there will be less opportunity for blood or secretions to enter the larynx. Patients undergoing operation on the throat can control the pharyngeal reflexes by relaxing and breathing through the mouth. The tongue should not be protruded. If the tongue is depressed downward and backward by using the depressor on the anterior portion of the tongue, instead of on the posterior portion, much less tendency for lack of control is occasioned. For injecting the anesthetic solution, a needle of small caliber at an angle of 45 degrees is satisfactory. Patients make almost no complaint of the insertion of the needle if the point is sharp. Both sides may be infiltrated before the operation is started. A solution of 0.2 per cent cocaine (or 2 per cent procaine) prepared with 0.5 cc. of 1:1000 adrenalin chloride solution to the ounce, and used for local infiltration, has proved effective in our hands. The first injection is directed into the posterior pillar

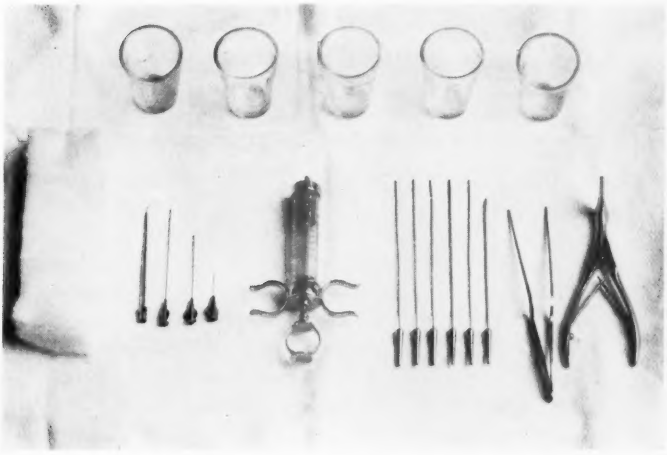


Fig. 2. Instruments and materials used in producing anesthesia for nasal operations.

between the so-called capsule of the tonsil and the aponeurosis of the superior constrictor muscles of the pharynx at the highest point (Figs. 1a, b and c). The injection is made slowly, so that infiltration, rather than the formation of a bleb, will result. If the injection does not proceed through to the lowest part of the pillar, it should be followed down. The next infiltration is made in the uppermost part of the anterior pillar. In piercing the pillar the surgeon must feel that the needle is perfectly free. The same slow infiltration as has been used previously is preferable and it will be found to extend along the pillar to the edge of the tongue. The next infiltration is made in the plica triangularis. This is accomplished by changing the direction of the needle toward, and longitudinally with, the edge of the tongue. The lower pole of the tonsillar fossa is more shallow than the upper pole; therefore, there is greater danger of penetrating the aponeurosis and causing deep cervical infection than is the case with the upper pole. There is no need of injecting the tonsil itself. Infiltration at this point is carried slowly around the lower pole.

The tonsil may now be seized with any suitable forceps and pulled rather strongly toward the median line and down toward the tongue. This outlines the upper pole. The needle is inserted

into the supratonsillar fossa at the margin of the tonsil and mucous membrane. The injection of the solution is carried on slowly, so that infiltration, rather than tumefaction, will take place. It might be added that as much solution may be used as is necessary to surround the tonsil satisfactorily because practically none of the fluid will be left after the tonsil has been removed. Previous peritonsillar inflammation may interfere with the infiltration because in such a circumstance the loose tissue will have been replaced by scar tissue. In such instances, infiltration may be repeated as often as necessary when the limits of infiltration are encountered. It is particularly important that the injection of the upper pole be carried on carefully, for unless the surgeon knows the position of the upper pole, the landmarks may be obscured and considerable difficulty will then be encountered in enucleation of the tonsil. The point of entrance of the last needle is a constant landmark. The patient is now ready for tonsillectomy.

Technic of Anesthesia for Intranasal Operations: Preliminary anesthesia of the nasal cavity can be effected in one of several ways. Spraying a weak solution of a cocaine derivative into the nose causes sufficient shrinkage of the membranes so that adequate visualization will be possible. Another method, just as effective, is to dip a cotton tampon in the anesthetizing solution, remove the excess of fluid rather well by squeezing the tampon between the fingers, and placing it between the turbinates and the septum, allowing it to remain in position for a short time. Trauma to the membrane should be carefully avoided, in order that normal recovery may take place. If the membranes are traumatized, they may be seriously damaged, convalescence of the patient may be unduly protracted and the normal physiologic activity of the membrane may be interfered with.

A tray on which the following instruments and solutions are placed has been found convenient for use (Fig. 2).

(1) Four or more pliable metal applicators.
(2) Metal nasal speculum.
(3) Bayonet-type nasal forceps.
(4) Glass or metal syringe of 2 or 5 cc. capacity.
(5) Needles for injection of solution of procaine hydrochloride.
(6) Two to 5 cc. of 10 per cent solution of cocaine.
(7) Two to 5 grains of cocaine powder or flakes.
(8) From 2 to 5 cc. of 1:1000 solution of adrenalin chloride.
(9) From 5 to 10 cc. of 1 or 2 per cent solution of procaine hydrochloride.
(10) Antiseptic solution.

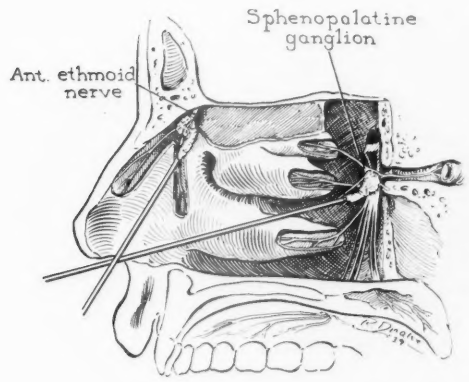


Fig. 3. The cotton-wrapped metal applicator in position over the naso-ciliary nerve and sphenopalatine ganglion.

If the preliminary procedure has been carried out with gentleness, the patient's confidence will be increased markedly, since from this point onward chances for painful manipulation become decreased with each maneuver. The greater part of the nasal mucous membrane is innervated by fibers from the sphenopalatine ganglion. The ganglion is situated deeply in the upper part of the pterygopalatine fossa, close to the sphenopalatine foramen. On removal of the cotton tampons, the nasal membranes appear shrunken and pale, permitting visualization of the postnasal space and facilitating the approach to the ganglion.

To anesthetize the ganglion[5, 6] a small amount of cotton is wound on the end of the metal applicator, then soaked in 1:1000 solution of adrenalin chloride, pressed relatively dry, and next dipped into the cocaine flakes. The flakes adhere to the moist cotton but do not penetrate the solution. This mixture has been called "cocaine mud." The applicator is passed into the nose at an angle between the posterior third portion of the middle turbinate bone and the septum (Fig. 3) until it reaches the posterolateral pharyngeal wall, where it is held or left in place for two to five minutes. The naso-ciliary nerve, from the ophthalmic branch of the trigeminal cranial nerve, supplies the sensory fibers for the mucous membrane of the anterior part of the nasal septum and the anterior part of the lateral

wall of the nasal cavity. For anesthetization of this region an applicator prepared in the manner described is placed in the cleft of the nose at a point between the attachment of the anterior end of the middle turbinate bone and the septum (Fig. 3). The applicator is left in place for two to five minutes. Operations for nasal polyps, on the turbinate bones, sphenoidal sinus, and for occlusion of the choana may be performed under such anesthesia. If the nasal septum is to be operated on, a small amount of a solution of 1 or 2 per cent procaine hydrochloride should be injected into the mucocutaneous junction at the site of the initial incision, in addition to the basic anesthesia. This procedure facilitates separation of the mucoperichondrium by hydraulic dissection.

Surgical procedures for operations on the paranasal sinuses by the intranasal route require more complete anesthesia of the structures than that which has been described. For such procedures, the maxillary branch of the trigeminal or fifth cranial nerve is injected with procaine hydrochloride. This may be accomplished by either the external route or the intra-oral route.[1, 3] Long, thin needles facilitate the procedure.

For the intra-oral route, the cheek is retracted upward and backward with the forefinger, and the needle is inserted into the mucous reflection above the first molar tooth, from which position it is advanced backward, upward and inward (in a plane transecting the inner canthus of the eye on the same side) 2.5 to 3.5 cm. from its point of entrance. Two or 3 cc. of a solution of 1 or 2 per cent procaine hydrochloride is now injected into the region, care being taken that the plunger first is retracted to avoid injecting the solution directly into a vessel.

For the external approach, the zygomatic bone is first palpated. The palpating finger is then moved below the lower border of the zygoma, and the patient is asked to open and close his mouth. This maneuver helps the anesthetist to form a clear mental picture of the relationship of the coronoid process to the zygomatic bone. A small wheal is raised on the skin of the cheek 1 cm. anterior to the coronoid process (Fig. 4). A flexible, 50 mm., 23-gauge needle is passed through the wheal on the skin and is directed slightly upward and backward until the point of the needle rests on the lateral pterygoid plate (Fig. 4). The needle is then slightly withdrawn and the tip of the needle is redirected anteriorly until is passes 0.5 cm. beyond the anterior border of the lateral pterygoid plate. The point of the needle now rests in the pterygomaxillary fissure and just in front of

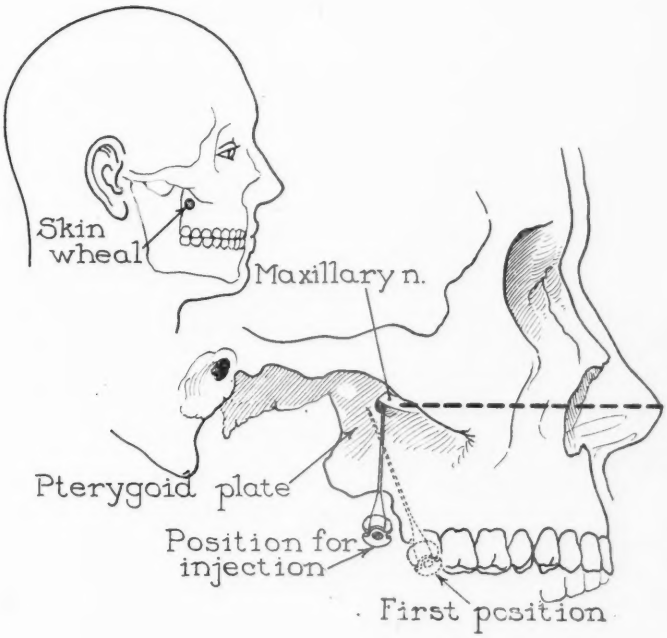


Fig. 4. Injection of the second division of the trigeminal nerve.

the foramen rotundum. This point is almost directly posterior to the tip of the nose. The syringe is now attached to the needle and gentle aspiration is done to make sure that the tip of the needle is not lying within the lumen of a vessel. Two and one-half to 3 cc. of a 2 per cent procaine solution containing adrenalin is deposited at this point.

An occasional paralysis of the sixth cranial nerve is noted following blocking of the second division of the trigeminal nerve. This may happen often if the intra-oral method is used. However, this abducent paralysis is transient, and while it is sometimes alarming to the patient, it should be of no consequence.

We believe that the extra-oral approach should be used when possible, for it is less painful to the patient than other methods. The needle can be passed through a sterile field and the bony landmarks

are so situated that no trouble should be encountered in depositing the anesthetic solution in direct proximity to the foramen rotundum, avoiding abducent paralysis. The intra-oral route might best be used if the patient has dermatitis, ulceration or lesions of the skin of any type on the face.

For the Caldwell-Luc operation, in addition, a solution of 1 or 2 per cent procaine hydrochloride should be injected along the labio-buccal fold at the site of the initial incision.

Anesthesia for the external frontal approach in operations on the frontal and ethmoidal sinuses is obtained in the same manner as that described for the intranasal method. In addition, however, a solution of procaine hydrochloride should be injected to produce an external block of the branches of the orbital nerve from the ophthalmic division of the trigeminal cranial nerve. To accomplish this, a solution of procaine hydrochloride should be injected into the skin along the supra-orbital margin medial to the supra-orbital foramen and along the inferior orbital margin medial to the infra-orbital foramen. The supratrochlear and infratrochlear nerves are anesthetized by means of the medial orbital block. This procedure is performed with a straight needle, which is inserted 1 cm. above the inner canthus of the eye and is pushed posteriorly 2 cm. and kept in contact with the medial bony surface of the orbital cavity. Executed in this manner, a minimal pressure is exerted on the orbital structures and a minimal distortion effect is caused, stripping of the periosteum is facilitated by hydraulic dissection, and local hemostasis is greatly increased. A paralysis of the third cranial nerve occasionally will be noted following this procedure. This paralysis is transient and the patient should be notified before the injection is made that a temporary double vision may develop. Care should be taken that only a small amount of solution is injected into the orbit, for anesthesia of the optic nerve, although it is usually of no consequence, is alarming to the patient.

SUMMARY AND CONCLUSIONS

1. Local anesthesia, with few exceptions, can be used successfully for the majority of patients submitted to tonsillectomy, intranasal operations, submucous resection of the nasal septum, or operations involving the paranasal sinuses.

2. It has been found that patients may be divided empirically into four groups in so far as their emotional reactions are concerned.

Recognition by the surgeon of these groups aids greatly in the conduct of the operative procedure.

3. Methods of producing local anesthesia in the aforementioned surgical procedures are presented.

4. A method combating the toxic effects of cocaine derivatives is described.

REFERENCES

1. Dogliotti, A. M.: Trattato di Anestesia; Narcosi—Anestesie Locali, Regionali, Spinali. Torino, Unione Tipografico Editrice Torinese, 313:3-4, 1935.

2. Knoefel, P. K., Herwick, R. P., and Loevenhart, A. S.: The Prevention of Acute Intoxication from Local Anesthetics. J. Pharmacol. & Exper. Therap., 39:397-411 (Aug.), 1930.

3. Labot, G.: Regional Anesthesia: Its Technic and Clinical Application. Second edition. W. B. Saunders Company, Philadelphia, pp. 75-148, 1928.

4. Lillie, H. I.: Certain Features of Local Anesthesia for Tonsillectomy. S. Clin. North America, 9:883-891 (Aug.), 1929.

5. Painter, A. M.: Cocain Anesthesia for Submucous Resection of Nasal Septum. J. A. M. A., 66:114 (Jan. 8), 1916.

6. Sluder, Greenfield: The Role of the Sphenopalatine or Meckel's Ganglion in Nasal Headaches. New York M. J., 87:989-990 (May 23), 1908.

7. Wetmore, F. H.: The Prolonged Toxic Effects of Local Anesthesia—Cocaine, Novocaine, and Allied Drugs; Untoward Effects of Nembutal. Canad. M. A. J., 34:299-300 (Mar.), 1936.

III

CHRONIC PROGRESSIVE DEAFNESS WITH SPECIAL REFERENCE TO ESTROGENIC SUBSTANCES

A Further Contribution*

Grant Selfridge, M.D.

San Francisco

In his discussion of the otosclerosis problem, the late Albert Gray,[1] speaking of the several theories, says: "There is an element of unconscious humor about some, as for example, that which attributes the disease to a deficiency of an ovarian hormone, the male human subject having apparently no existence in the scheme of things." Further on he says: "Women are more frequently the subjects of otosclerosis than men, the relationship being forty per cent men to sixty per cent women. The usual explanation for this preponderance of women is that the ovarian hormone or hormones act in some way upon the metabolism of bone either directly or indirectly." In his summary he says: "The essential causative factor of otosclerosis is a gradually increasing defect in the vasomotor mechanism which governs the nutrition of the organ of hearing as a whole, and there is no evidence whatever of any defect in any of the endocrine glands nor any evidence of any defect in the bone metabolism of the body."

This excellent article of Doctor Gray's appeared in the Journal of Laryngology and Otology in October, 1934, not long before his untimely death. Had he lived longer, he no doubt would have corrected his views, because the literature appearing since offers a satisfactory explanation for many of the statements made in his article.

It is therefore my purpose to discuss these several points and to show in several clinical histories with audiometric charts the part the sex glands and at least two factors of the vitamin B complex play, not only in certain ear conditions but also in improving the general health and well-being of this group of unfortunate human beings (subjects of chronic progressive deafness).

---

*From the Harriman Research Laboratories, Southern Pacific General Hospital.

RELATION TO SEX GLANDS

Langdon Brown[2] says, "The male and female hormones are closely related chemically" and "At first sight, it seems extraordinary that a female hormone should have a masculinizing effect." McLellan[3] thinks that boys who respond to this treatment may, during intra-uterine life, have been subject to an excessive amount of estrin or to a deficient amount of anterior pituitary substance.

Both male and female substances are found in the blood and urine of pregnant animals (mares) and in humans. Collip[4] has recovered them from the placenta. Thompson and Heckel[5] say: "Rapidly accumulating evidence clearly shows that the anterior pituitary-like principle from the urine of pregnant women exerts a profound influence on the growth and function of the male genetalia . . . before the age of puberty." Likewise it is well known that estrogen has a marked influence on the development of the female sex characteristics.

Many abnormal sex behaviors seem explainable on the basis of excessive hormonal substances found in the blood at different periods. Many males seem to have increased ovarian rests, and females have excessive maculinizing rests, to explain variations in body configurations, sex responses, etc.

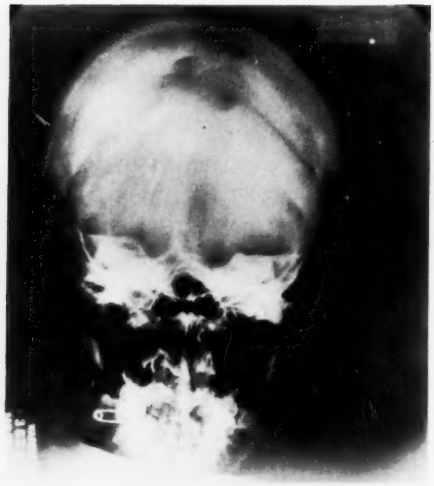
It is therefore quite conceivable that during fetal growth there may be a lack of normal balance between the androgenic and estrogenic substances which is evidenced after birth and often discoverable around the puberty period, particularly in an otosclerotic family where the mother may have frank evidence of endocrine dysplasia, coupled with faulty nutrition and a lack of several of the growth-promoting factors including certain of the vitamins.

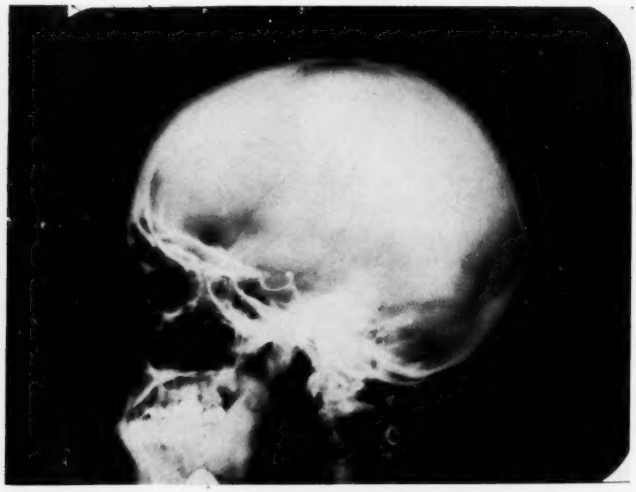
More careful study of masculine deafness along these and allied chemical and endocrine lines offers (to my mind at least) a satisfactory explanation for the preponderance of otosclerosis in females.

WHY ARE MORE WOMEN DEAF?

Gray[1] justly says, "At every menstrual epoch a profound disturbance takes place in the vasomotor mechanism of the organs of reproduction in woman." There is no doubt that women as a result of this are subject to more emotional stress and strain and are so often classified as vasomotor unstables.

Quite recently Novak[6] in discussing the vasomotor variations and symptoms occurring during the menopausal period (and this,





These pictures show the increased density of the skull and are from a woman, aged forty years, diagnosed by an endocrinologist as a hypopituarism with girdle fat, hypertricoses, irregular catamenia and mild hypothyroidism. Among other things, she was given parathormone to which she became refractory, which probably explains the excess calcium of the skull, pelvis, and elsewhere.

I think, also applies to the puberty period) mentions the "existence of a rhythmic sex center * * * in the parahypophyseal area which is quite certainly hooked-up with the hypophysis itself and offers the logical hint as to the location of the psycho-neuro-hormol liason concerned in the vasomotor disturbances in these periods." Langdon Brown[2] mentions that "the diencephalon has ties in three directions: with the cortex of the brain above; with the pituitary above; and with the sympathetic system. In this way, a psychical experience may record its effect either through the chemical mechanisms initiated through the pituitary, or through the sympathetic system acting on the viscera or on the other ductless glands!"

It is not surprising that the first marked loss of hearing is definitely related to the puberty period if, with the above mentioned anatomical peculiarity, there is an hereditary tendency toward deficiency of certain of the endocrine glands (pituitary, thyroid, or ovaries) as is so frequently found together with disturbance is calcium, phosphorus, and magnesium metabolism and also coupled with a definite evidence of disturbances of nutrition, as shown in the excessive use of carbohydrates, lack of milk, eggs and leafy salads, and little or no citrus fruits and vitamins (especially the vitamin B complex).

My own observation of a series of cases studied in recent months is that the majority give definite evidence of the need for estrogenic substances, i. e., scanty or shortened menstrual epochs, marked nervous and emotional episodes, etc. Most of these individuals show a lowered basal metabolic rate and my experience is that the use of estrogenic substances before the addition of thyroid often brings about a very definite improvement in the impaired menstrual functions, nervousness, fears, fatigue, tinnitus (roaring like a sea shell), together with some improvement in the hearing curve.

In relation to lowered basal metabolic rates, recent work indicates that many are undoubtedly of nutritional origin and depend on lack of sufficient protein. Hayden[7] has called attention to very low basal metabolic rates in pernicious anemia. Some of his cases looking like hypothyroids had basal metabolic rates which became normal after the correction of nutritional deficiencies. Speeding up of the metabolic processes by whatever process—protein, thyroid, estrogen, etc.—through their decalcifying action, offers to my mind the only explanation of improvement in the low tones when it occurs.

THE RELATION OF PUBERTY AND OTHER FACTORS TO BONE CHANGES

There seems to be no doubt that in a very large number of cases there is a very definite relationship at puberty to well-pronounced beginning deafness and the closure of the epiphyses. This applies only to otosclerosis and appears to be related to increased deposition of calcium. In adhesive deafness the primary fault appears to be due to unresorbed mesenchyme and that is related to lack of cell cementum, i. e., vitamin C.

Mortimer, Collip and Wright[8] have called attention to cranial dysplasias occurring in atrophic rhinitis and have suggested similar findings in otosclerosis. In a number of my cases, x-ray studies of the calvarium, petrous bones and the pelvis showed increased calcium deposit.

In relation to this, it is a well-known fact that hyperactivity of certain endocrines (pituitary, thyroid, parathyroid, adrenal cortex, gonads) produces a decalcification of bones, whereas hypoactivity brings about increased calcification. In rats deficient in magnesium in the early stages there is a marked increase in deposit of calcium.

Burrows[9] reports the use of Hanson's and also Collip's parathormone extract and shows that in many animals the extracts become refractory and a condition known as marble bone results.

That the vitamins play an important role in calcification and decalcification is beyond question. The recent paper at the Congress of Physiologists (Zurich, August, 1938) by Mellanby[10] tells of his finding in young animals deficient in vitamin A increased deposit of bone in the modiolus with pressure destruction of the eighth nerve. He also found increased calcium elsewhere in the skeleton.

Nothing is known as to the role of the vitamin B complex so far as calcification and decalcification are concerned but there is some evidence that in Jukes' dermatitis chick deficiency (a filtrate factor of the vitamin B complex) the bones are harder than normal. Studies in relation to bone density in the different fractions of the B complex are being conducted with the spectroscope and subsequently these findings will be confirmed or disproved by bone section study with the microscope.

Abt[11] quotes Aub, saying: "Vitamin C is a factor in the calcium deposition in bones. Low blood ascorbic acid and rarefaction of the alveolar bone has been noted."

Further, Rinehardt[12] has already reported in his C deficient guinea pigs decalcification in certain of the long bones and Ono[13] has found even the ossicles partly decalcified in C deficiency animals. Vitamin C is the intra-cellular cementum, and its lack probably explains unresorbed mesenchyme. During fetal development its lack may have a far-reaching effect, more perhaps than is visualized at this time. A few of our cases show a low C in the blood at ages as late as thirty-five and forty-two years. A questionnaire to sixty members of the San Francisco League of the Hard of Hearing showed that they were all deficient in vitamin C, most, if not all, never ate an orange in childhood, and over fifty per cent gave a history of very poor teeth in childhood and adult life.

Agnes Fay Morgan's experimental use of D_2 (calciferol) and D_3 is extremely interesting. She found extensive decalcification with D_2, but not so great when D_3 was given. In addition to the bone decalcification in these dogs, there were found extensive calcium deposits in the soft tissues, kidney, heart, aorta, and especially in the lungs and in the muscle sheath and tendons. Also, she found in the study of the urine of her dogs a picture about identical to Bright's disease in humans.

It is well known that dietary deficiencies in animals produce bone softening. This has been pointed out by several writers and particularly by Weber and Becks.[14] It has been produced by drugs in humans and corrected by diet as Gill[15] has reported. Even in von Recklinghausen's disease, the bone absorption has failed of correction, in spite of the removal of the parathyroid glands and was markedly relieved by dietary measures.

It is also well known that dietary deficiencies in the pregnant mother will explain poor teeth in the baby, and as the teeth form about the same time as the petrous bones, it is not surprising that an otosclerotic process may have its beginning in fetal life. Minot[16] said recently: "It is well to remember that since the nutritional states of the mothers affect the well-being of infants, the health of the whose population depends to a greater or lesser extent on maternal nutrition. Likewise let us recall that at no period of life is optimal nutrition more important than in infancy and childhood because nutritional defects so readily produced then may be at the root of disorder arising at any time later in life."

The following chart indicates the scope of the studies carried on during the past seventeen months, and seems to show in part that the various factors influencing the vasomotor system, combined with

nutritional deficiencies, initiate the beginnings, and that the addition of these "little things of life" (endocrines, mineral salts, vitamins, etc.) mentioned by Barnett Sure[17] play a dominant part in either improving the hearing or arresting the progress of the deafness.

THIAMIN CHLORIDE AND NICOTINIC ACID

The exact role of vitamin B in the cases under discussion in this paper is not clearly understood, but we must accept the truth as expressed by McCarrison,[18] who says that the B complex affects every part of the body, the hair, skin and nails, the muscular and bony systems, the general and autonomic nervous system, the cardiovascular system, and the mucous membranes, particularly the intestinal tract.

The very fact that in about two hundred cases of both nerve and conduction deafness the urine shows that they are all deficient in the output of thiamin chloride (B_1) argues that the dietary intake is either deficient in that factor or else the intestine through long disuse fails to pick out the substance from the regular diet. It is the experience of more than one investigator that when it is used parenterally the urine in a varying time shows a definite increase, whereas it often does not show a similar increase when given by mouth in substantial amounts.

As to nicotinic acid in conduction deafness, one cannot state definitely how it acts. Quite recently Spies[19] in a personal communication said: "When sufficient quantities of nicotinic acid are administered to a pellagrin, the concentration of cozymase in his blood and urine rises to normal levels. You will remember that cozymase is one of the fundamental enzymes in cell life. It is composed of the amide of nicotinic acid, ribose, phosphoric acid and adenine."

It is also a vaso-dilator substance as observed in many individuals, producing a marked flushing of the face shortly after taking varying doses. It is quite possible that it has also through this vaso-dilator response, a decalcifying property similar to that occurring in certain endocrine states previously mentioned.

Recent unpublished information about the histopathological studies by Dr. Jesse L. Carr of the Morgan gray haired rats deficient in the filtrate factor, indicates that this factor is probably most important in senescence, not only of animals but also of humans. These studies indicate a definite atrophy of the adrenal cortex, the medulla, gonads, skin, and loss of melanin, etc. Injections of corti-

cal substance, in spite of the continued deficiency, restores the gray hair to normal. Histological studies of the cord and the eighth nerve have not yet been completed.

The inspiration for this study followed a hint given by Collip and the story up to this point was written in May, 1938. Quite recently, Mortimer, Wright, Thompson and Collip[20] have published their article on constitutional deafness. They investigated the cranial skiagrams in one hundred and fifty-three patients of whom ninety-four were females and fifty-nine males. They state that the "cranial type is a reliable index of its constitutional type" and show a definite dysplasia. "The majority of women show the hypofunctional aspect of dyspituitarism in the ascendant (75%)." This shows itself in "sclerosis of the calvarium and by a degenerative structural change." They also state: "There are, however, in both groups (atrophic rhinitis and constitutional deafness) a significant number of women who show evidence of pituitary hyperfunction in the years of growth, and this is slightly more apt to be the case in constitutional deafness than in atrophic rhinitis * * * in males this statement is still more justified, for in both diseases about forty per cent show an instability on a hyperfunctional side. Sclerosis, indicative of degenerative change, is much less marked than in the females." In the last portion of their summary, i. e., number four, they say: "It is shown that treatment which acts specifically upon the nasal disease (atrophic rhinitis) is capable of producing not only marked improvement of the aural defect in certain individual cases but also a statistically significant amelioration in the hearing level of a group of constitutionally deaf as a whole."

My own studies correspond somewhat to the findings of the McGill group, except that I have not observed the percentage of dyspituitarism, nor have I obtained the high percentage gain shown in several of their cases. Many of my cases show a low basal metabolic rate, and my feeling is that thyroid plays perhaps a more important role than estrogens in changing the character of the new bone at the foot-plate of the stapes. Unless such change takes place, it is impossible to believe that improvement for low tones is otherwise likely. My use of estrogenic substances* was by mouth and parenterally. It is quite possible had I used the estrogens intranasally and for a longer period of time—one to two years—the results might have been more in line with those of the Canadian reporters. Apparently, through a better interpretation of their x-ray plates, more

*These substances were furnished in very substantial quantity by Dr. Max Gilbert of the Schering Corporation, Bloomfield, N. J.

CHART

Names	Age	B.M.R.	Hgb.	Vitamin B_1	Porphyrin	Tinnitus	Catamenia
Miss M. D.	17	—6	127	16.0	Negative	Occ. buzzing	Painful and scanty
Miss K. H.	19	—9	86	12.6	Plus one	Sea shell	Cramps and irregular
Miss V. R.	24	—21	90	11.9	Plus one	Occ. buzzing	Profuse
Mrs. J. L.	25	—12.9	90	11.2	Trace	Ringing	Profuse
Miss G. O'B.	30	—1	87	16.0	Negative	Sea shell	Painful
Mrs. E. G.	31	—9	72	7.0	Plus one	Roaring	Cramps and dizziness
Miss D. N.	32	—12	85	5.0	Plus two	Like steam	Cramps and irregular
Mrs. R. L.	34	—17	(not done)	16.5	Negative	Roaring	Surgical menopause
Mrs. B. R.	35	—16	100	3.0	Negative	Roaring	Normal
Mrs. A. Ahl.	36	—12	92	5.1	Negative	Occ. buzzing	Profuse and cramps
Mrs. B. H.	36	—10	81	9.5	Plus one	Sea shell	Scanty
Mrs. F. E.	39	—1	86	15.0	Negative	Sea shell	Irregular
Mrs. A. F.	41	—1	78	6.8	Negative	Pulse-like	Beginning climacteric
Mrs. T. M.	41	—14	62	13.5	Negative	Roaring	Scanty
Mrs. C. O'S.	43	—12	(not done)	10.8	Plus one	None	Beginning climacteric
Mrs. B. P.	50	—9	(not done)	14.4	Negative	None	Scanty
Mrs. R. McN.	51	—20	83	18.2	Negative	Pulse-like	Menopause 1 year ago
Mrs. A. T.	57	(not done)	82	15.0	Negative	None	Menopause years ago

evidence of pituitary disturbances were found in their cases. The improvement in hearing of their cases resulted probably from activating the pituitary by the use of estrogenic substances.

It will be observed in my charts that most cases show a low basal metabolic rate and also a low B_1 output in the urine as well as increased porphyrin. The cases studied so far for the nitrogen caloric ratio show deficient protein intake. While I tried estrogenic substances primarily for its effect on hearing and the tinnitus, my one concern was to relieve if possible the menstrual difficulties of which they so often bitterly complained. The recent study of Foster and Thornton[21] regarding the use of thyroid in menstrual difficulties shows that estrogenic substances do not always correct the faults present, and Jones, in his studies, has suggested that lack of sufficient protein has a definite influence on the complaints occurring during the menstrual epoch. His statements may require a long period of trial before they are confirmed or disproved. It seems evident from Fluhmann's writing and that of other workers in the field of Gynecology, that these estrogenic preparations have to be used in many cases recurrently over a long period of time.

I was satisfied in the early studies, and am now more than ever convinced that, while constitutional deafness (otosclerosis) is a problem occurring during the stage of greatest sexual activity (between eighteen and thirty-five years) and that while the subject of sex is the dominating thing in life, the problem is not one of sex alone, but is one involving not only the endocrines but also minerals, vitamins and proteins. In other words, it involves all the factors engaged in the metabolism of growth, their hereditary and environmental factors, and in the final analysis resolves itself into a nutritional deficiency.

CLINICAL HISTORIES

CASE 1.—Miss Jean F., age 15. January 7, 1938.

Complaints: Deafness—left ear.

History: Deafness: Noticed deafness in left ear three years ago. No family history of deafness. Tinnitus: occasional buzzing.

Allergy: No history of hay fever or asthma. Has had hives.

Teeth: Good.

Diet: Difficult to obtain satisfactory dietary history (prenatal) from mother. Diet of patient up to the age of seven was perfectly balanced. After that age, her diet contained too much starch and sugar. Patient is now on a reducing but balanced diet.

Diseases: Whooping cough, measles, mumps.

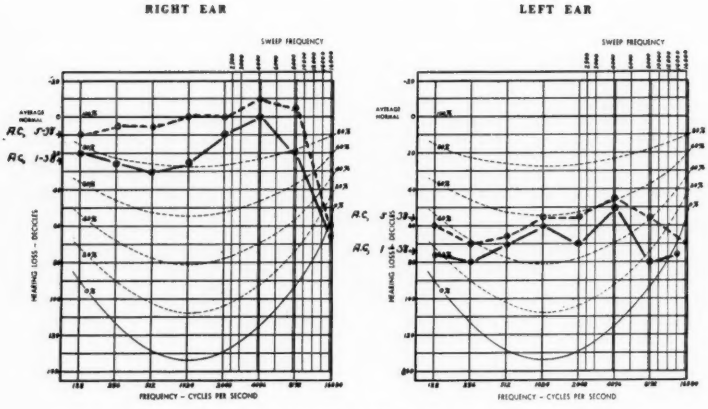


Fig. 1, Case 1.

Operations: Tonsils removed.

Catamenia: Began at age of twelve. Not regular.

Examination: Physical—Done by Dr. Myrl Morris. Patient has gained 22 pounds since April, 1937. She has a typical pituitary dyscrasia with only a very slight thyroid involvement. There is a very great breast development, but the nipple is very small and immature. In the genital area the clitoris is very immature and the labia are only slightly developed. Uterus is small and ovaries thought to be normal. There is the typical distribution of fat around the abdomen and the hips with an even distribution through the thighs and legs.

Weight: 156 pounds.

Height: 5 feet 1 inch.

Blood Pressure: 106/58. (Heart and lungs also normal.)

Nose: Normal.

Sinuses: Normal.

Tonsils: Out.

Ears: Right, drum normal; left, light spot broken.

Tubes: Normal.

Hair: Fine and oily. Eyebrows typically pituitary.

Laboratory Data:

B. M. R.: Minus 4%.

Blood Count: Hgb. 92%; R.B.C. 4,100,000; W.B.C. 5200.

Blood Sugar: (Micro method) 162 (½ hr.); 130 (1 hr); 107 (2 hr.).

Blood Analysis: Calcium 12.5%; Phosphorus 3.7%.

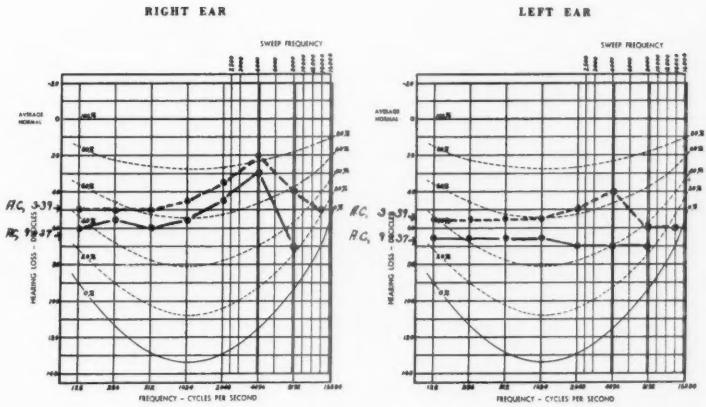


Fig. 2, Case 2.

X-rays: The frontal sinuses are not developed. The other paranasal sinuses and the mastoids are well developed. The face is small in proportion to the size of the skull. The skull shows a normal amount of calcification and is of normal thickness. The sella is small and flattened. The findings are suggestive of a mild hypopituitarism. Petrous bones show some calcium increase.

Treatment: January 7, 1938: 1) To have 20 injections of antuitrin 'S'—one twice a week; 2) whole gland pituitary, grains six daily; 3) thyroid, one-half grain daily; and 4) given a reducing diet.

March 24, 1938: Progynon tablets, 200 rat units daily, and to continue the pituitary, thyroid and reducing diet.

May 27, 1938: Patient discontinued all treatment.

Comments: On March 24, 1938, patient's hearing had improved in both ears. On May 27, 1938, following the use of Progynon for two months, there was an additional improvement in the hearing of the left ear. Continued treatment over another year might have shown further gain in the hearing curve of the left ear.

Case 2.—Miss Minerva D., age 16. July 29, 1937.

Complaints: Deafness—both ears.

History: Deafness: Noticed deafness six years ago; very marked when fourteen years old. No history of deafness in family. Tinnitus: occasional roaring and buzzing. Had abscessed ears when eleven months old.

Allergy: No history of hay fever or asthma.

Teeth: History of poor teeth in childhood. Mother's tooth history and dietary intake are not good—probably had much to do with condition of child's teeth.

Diet: In childhood, dietary intake was not good, especially the vitamin C intake. At the present time her dietary habits seem to be quite satisfactory.

Diseases: Chickenpox at five; measles at six; German measles at fourteen.

Operations: Tonsils out at age of five.

Catamenia: Began at age of twelve. Periods are regular, lasting three days. Are accompanied by extreme nervousness. The first day she has nausea with vomiting and pains. Has to stay in bed for twenty-four hours, missing one day of school each month.

Examination: Physical—General health very good except that she catches cold easily—four or five head colds a year.

Weight: 115 pounds.

Height: 5 feet 6 inches.

Nose: Slightly high deviation of septum.

Tonsils: Out.

Ears: Normal in appearance.

Laboratory Data:

B. M. R.: Minus 3.3%.

Blood Count: Hgb. 127%; R.B.C. 5,790,000; W.B.C. 8050.

Blood Analysis: Cholesterol high; phosphatase low; calcium, phosphorus, sodium and potassium are normal; R.B.C. magnesium is slightly high.

X-rays: The frontal sinuses are well developed and are within normal limits. The mastoids and other paranasal sinuses are well developed. The size of the face is normal in relation to the size of the skull. There are rather prominent convolutional markings on the inner table of the skull in the frontal and parietal regions. The skull shows normal calcium content, except the petrous bones. Conclusion: the findings are within normal limits, but there is a suggestion of hyperpituitarism. Bone age corresponds to eighteen years.

Treatment: July 29, 1937: Thyroid, 1 grain daily (to September 18, 1937, at which date pulse reached 100 and there was some loss of weight; to be continued later).

December 9, 1937: Estrogenic substances (to November 16, 1938).

September 19, 1938: Sodium nicotinate capsules, 4 grains daily (to November 16, 1938).

November 16, 1938: Nicotinic acid, grains 1, and B_1, 1000 I.U., one capsule night and morning (to March 30, 1939).

March 30, 1939: To use estrogenic substances again.

Comments: Patient has had complete relief from nausea, tinnitus, etc., while taking estrogenic substances; recurrence of symptoms in less severe form followed stopping of the medication. The improvement in the hearing curves of both ears appears to be due to the use of these estrogenic substances. There was no further change in hearing following the use of nicotinic acid and B_1.

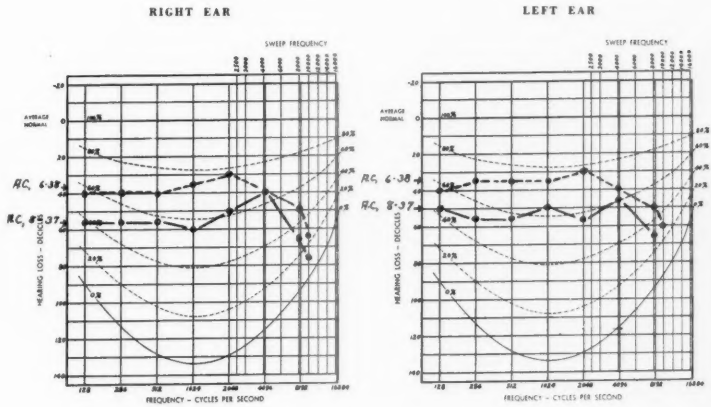


Fig. 3, Case 3.

CASE 3.—Miss Floride H., age 21. November 21, 1936.

Complaints: Deafness—both ears.

History: Deafness: Noticed deafness at age of fourteen, following colds. Had earache and abscessed ear in childhood. Hears better in noise. No deafness in family except a grandmother whose deafness followed scarlet fever. Father has slight deafness due to gun practice in U. S. Navy. Tinnitus is intermittent; like the roar of a sea shell and is worse when patient is fatigued.

Allergy: No history of hay fever or asthma; occasional hives.

Diet: In childhood and adolescence, she was deficient in vitamins A, B, and C. Present history indicates that dietary habits are fair. (Grandmother, who is seventy-six and very hard of hearing, had a diet most of her life of meat and potatoes, with no fruit and very few vegetables. Mother's dietary history not obtainable, especially as it relates to the gestation period of patient. Presumably some of the grandmother's dietary faults passed on to the patient's mother—at least during her childhood and adolescence.)

Operations: Tonsils removed.

Catamenia: Began at age of twelve. Apparently was normal until age of eighteen, at which time patient was at Vassar College and began to have cramps the first day. No headaches, stuffy nose, or nausea. Periods since then have been pretty regular and fairly profuse.

Examination: Physical: General health good.

Nose: Normal. No postnasal discharge.

Tonsils: Out.

Sinuses: No evidence of sinus involvement.

Ears: Hearings tests—Conversation: R. 20″, L. 21′.
Whisper: R. 0″, L. 20″.

Tubes: Open.

Laboratory Data:

B. M. R.: Minus 4% (August 30, 1937); minus 1% (May 31, 1938).

Blood Count: Hgb. 84%; R.B.C. 4,430,000; W.B.C. 8800.

Sugar Tolerance: Low curve.

Blood Analysis: Phosphorus, sodium, potassium, cholesterol are normal. Calcium, R.B.C. magnesium and serum magnesium are high. Phosphatase is slightly low.

X-rays: There is a slight increase in calcium contents of the alveolar process and pelvic bones, but films of the skull and mastoids show somewhat diminished calcium content.

Treatment: December 9, 1936: Given the filtrate factor of the vitamin B complex (rice bran), 6 teaspoonsfuls daily (to May 15, 1937).

September 10, 1937: Magnesium carbonate and strontium carbonate, 15 grains each, t.i.d., for several weeks.

September 29, 1937: Given a series of injections of riboflavin (to February 7, 1938).

December 17, 1937: Progynon tablets, 200 units daily except during periods. Two weeks later the dose was increased to 600 units daily (to February 10, 1938).

February 10: 1938: Progynon tablets reduced to 400 units daily (to June 23, 1938).

June 23, 1938: Nicotinic amide, grains one-half daily (to August 10, 1938).

Comments: This patient shows a definite gain in air and bone conduction in both ears (see audiogram). She took Progynon from December 17, 1937 to June 23, 1938, and a greater part of the improvement in hearing was due apparently to the use of this estrogenic substance. The filtrate factor (that portion of the rice bran vitamin B complex from which B_1 riboflavin had been removed with Fuller's earth) may have played some part, but it was impossible to determine by audiometric tests as the patient was living near Los Angeles at that time. The value of the nicotinic amide is undetermined for the same reason.

Case 4.—Mrs. H. L. C., age 25. December 28, 1937.

Complaints: Deafness—right ear.

History: Deafness: Noticed deafness at the age of seventeen years. Onset followed colds. Had abscess in right ear in childhood. Patient's mother is quite deaf at age of forty-five; noticed signs of deafness at twenty-four. Brother is also slightly deaf. Patient has no tinnitus.

Allergy: No history of hay fever or asthma.

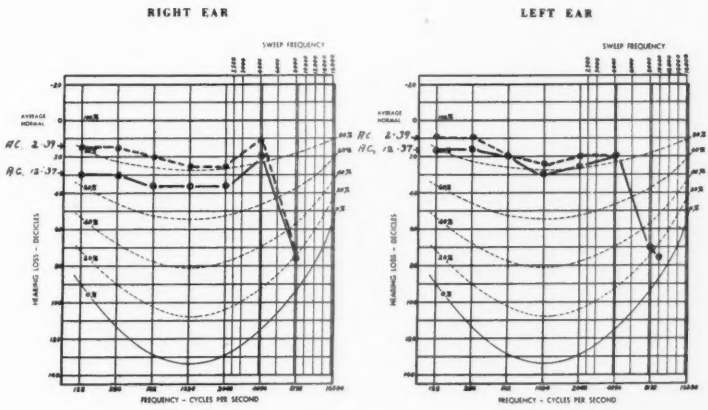


Fig. 4, Case 4.

Teeth: Went to dentist frequently in childhood. At present, has seven small silver fillings and upper wisdom teeth show decay.

Diet: Dietary history to date shows too much carbohydrates; little milk or eggs, and no citrus fruits. Diet low in all the vitamins, especially B and C.

Diseases: Smallpox and all other childhood diseases.

Operations: None.

Catamenia: Began at age of twelve. At present, a moderate degree of menorrhagia.

Examination: Physical: Done by Doctor Grant Ellis. Examination showed a normally developed but somewhat undernourished woman. Past history is essentially negative, except that patient, when examined, gave a history of loss of fourteen pounds during the preceding nine months. At present time has frequent colds.

Weight: 110 pounds.

Height: 5 feet 6 inches.

Blood Pressure: 120/75. (Lungs clear; heart not involved.)

Nose: Normal.

Thyroid: Palpable, but not enlarged.

Tonsils: Moderate size.

Ears: External canal and ear drums are normal.

Tubes: Normal. An adhesion to left tube.

Laboratory Data:

B. M. R.: Plus 3%.

Urine: Negative.

Blood Count: Hgb. 90%; R.B.C. 5,000,000; W.B.C. 10,150.

Blood Sugar: 90 (Fasting); 104 (½ hr.); 120 (2 hr.); 115 (3 hr.).

Blood Analysis: Phosphorus, potassium, R.B.C. and serum magnesium are normal. Calcium is slightly high; and sodium and phosphatase are slightly low.

X-rays: The frontal sinuses are well developed and extend lateralward in the supraorbital ridge. The ethmoids and antra are larger than normal. The petrous portions of the temporal bones and the floor of the anterior fossa show increased calcium content. The sella is large, measuring 15 mm. in its antero-posterior diameter. The clinoid processes are within normal limits. The calvarium is of normal thickness but shows increased calcium content. The tables of the skull are not differentiated. The face is slightly larger than one-half the size of the skull. These findings indicate a sclerotic skull superimposed on an early subacromegalic type.

Treatment: January 13, 1938: Given nicotinic acid capsules, grains one daily; and Progynon tablets, 400 rat units daily.

April 13, 1938: To continue taking nicotinic acid capsules, same dosage; and stop the Progynon tablets. To be given injections of A.P.L. twice a week.

June 13, 1938: To increase dosage of nicotinic acid capsules to two grains daily. Patient is to come in ten days before each menstrual period for an injection of A.P.L., and again five days later for a second injection.

October 14, 1938: Sodium nicotinate capsules, grains one, night and morning.

January 21, 1939: Nicotinic acid, grains one, and thiamin chloride, 1000 I.U., one night and morning.

Comments: This patient has shown a definite improvement throughout the entire tone scale of the right ear and most of the notes of the left from the use of A.P.L. (Antuitrin 'S') and nicotinic acid. Certain environmental factors had a bearing on this case. The strain and care of two children and the household work prevented her from getting sufficient rest, together with visits from her mother-in-law and other relatives with the accompanying irritations. When these conditions were relieved, fatigue and other symptoms were vastly improved.

Case 5.—Mrs. C. D. H., age 36. February 7, 1938.

Complaints: Deafness—left ear.

History: Deafness: Noticed deafness ten years ago. Worse after birth of child four years ago. Has tinnitus, roaring like a sea shell. Cause apparently not associated with colds.

Allergy: None found although she has had hives. Thoroughly tested by Dr. Rowe.

Teeth: Good until twenty-one years of age, when several were filled.

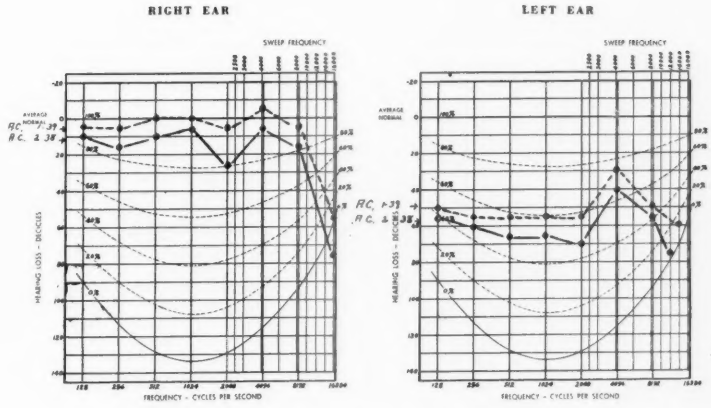


Fig. 5, Case 5.

Diet: In childhood and adolescence had less than average amount of milk; four to six eggs weekly; no citrus fruits until sixteen years old and then not regularly. Plenty of bread, potatoes and cereals; no salads; one or two vegetables. Meats (?). In adolescence, deficiency in vitamins A, B and C; probably D also. In adult period, had no milk, few eggs, few salads. Definitely deficient in B and C.

Diseases: Measles at age of six; pleurisy at age of twenty-three; no definite tuberculosis. Mild influenza at age of sixteen.

Operations: Tonsillectomy and appendectomy at age of twenty. Left tube and ovary out in 1933.

Catamenia: Began at age of thirteen. Now presents a mild ovarian deficiency.

Examination: Physical: Done by Dr. Lisser. Nothing of note except uterus enlarged. One or more fibroids. Mild hypothyroidism and easy fatiguability.

Weight: 148 pounds.

Height: 5 feet six inches.

Blood Pressure: 114/70.

Nose: Slight high deviation of septum. Posterior ends of middle turbinates whitish.

Sinuses: Normal.

Tonsils: Out.

Ears: Right drum normal except broken light spot. Left normal.

Tubes: Open.

Hair: Dry.

Nails: Brittle.

Laboratory Data:

B. M. R.: Minus 10% (February 12, 1938); plus 2% (December 7, 1938).

Urine: Few pus cells.

Blood Count: Hgb. 84%; R.B.C. 4,030,000; W.B.C. 4,600.

Blood Analysis: Calcium, phosphorus, sodium, potassium, R.B.C. magnesium are normal. Serum magnesium is 3.6%.

Vitamin C: 1.2%.

Vitamin B_1: 9.5 I.U./24 hours; porphyrin, plus one.

X-rays: The skull is moderately increased in thickness. There are convolutional markings of the inner table, most marked in the frontal area. The skull shows normal calcium content. The sella is normal. The frontal sinuses are normal in size. The facial bones are not out of proportion to the size of the skull.

Treatment: February 15, 1938: Given reducing diet; Progynon tablets, 200 rat units daily; and Armour's thyroid, one grain daily (to May 13, 1938).

April 4, 1938: Vitamin B syrup, 1 teaspoonful three times daily, was prescribed, but patient took its equivalent in tablet form instead (to May 13, 1938).

May 13, 1938: Continue with vitamin B tablets; Progynon tablets increased to 400 rat units daily; and thyroid increased to two grains daily (to September 23, 1938).

September 23, 1938: Continue Progynon tablets at 200 rat units daily; thyroid at same dosage; and an injection of thiamin chloride daily, 3000 I.U. per cc. (to November 4, 1938).

November 4, 1938: Progynon, same dosage; thyroid increased to three grains daily; and nicotinic acid capsules, three grains daily (to March 23, 1939).

March 23, 1939: Progynon, same dosage; thyroid reduced to two grains daily; and stop nicotinic acid capsules (to May 26, 1939).

May 26, 1939: Stop Progynon; continue thyroid, same dosage; Galen B syrup, 1 tablespoonful daily.

Comments: The results of treatment to date (May 26, 1939) have been gratifying. Her hearing is improved and her easy fatiguability has disappeared. It is impossible to state how much hearing was gained from the use of the Progynon because thyroid was ordered at the same time by her family physician. Nor is it possible to show how much improvement followed the use of thiamin chloride and nicotinic acid. However, the net result of fifteen months' treatment is shown in the audiogram. In addition to this, the tinnitus has been entirely relieved, her menstruation has been normal for several months, and the patient says that she hasn't felt so well in ten years and is quite conscious of the improvement in her hearing.

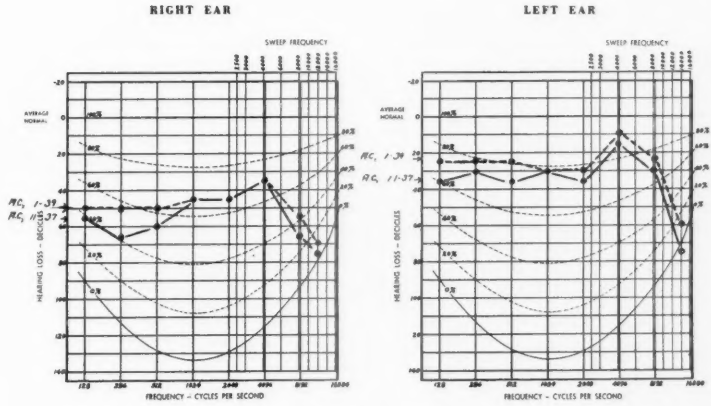


Fig. 6, Case 6.

CASE 6.—Mrs. T. J. M., age 40. November 1, 1937.

Complaints: Deafness—both ears.

History: Deafness: Noticed deafness in 1928. Deafness followed severe colds; had crackling sounds in ears. No ear abscesses. No family history of deafness. Tinnitus is of a roaring character—like a sea shell—and occasionally pulse beat. Hears better in noise.

Allergy: No history of hay fever, asthma, or hives.

Teeth: In childhood, teeth were not good. Had considerable dental work done about twelve years ago.

Diet: In childhood, diet was low in vitamins A, B and D; and was quite out of line (too much carbohydrates, etc.) until the past year or two ago. At present, diet is low in vitamin B.

Catamenia: Menopause started about one year ago.

Examination: Physical: General health is good. Does not catch cold easily. No vertigo, or migraine. Was constipated years ago, but not at present.

Weight: 142 pounds.

Height: Five feet 7½ inches.

Nose: Normal.

Sinuses: Normal.

Tonsils: Present; no obvious infection.

Ears: Slight retraction of drums. No movement anterior half.

Tubes: Open.

Laboratory Data:

B. M. R.: Minus 13.5%.

Blood Count: Hgb. 50%; R.B.C. 3,900,000; W.B.C. 6,400. R.B.C.'s show marked achromia; slight anisocytosis; polychromatophilia; and occasional stippled cell.

Vitamin B_1: 13.5 I.U./24 hours; porphyrin, negative.

Blood Analysis: Calcium, phosphorus, sodium, potassium, R.B.C. magnesium and cholesterol are normal. Phosphatase is low (slightly); and serum magnesium is slightly high.

X-rays: The calcium content of the base of the skull, pelvis and the alveolar process is somewhat above normal.

Treatment: November 7, 1937: To have liver injections every ten days; iron tablets; and an injection of theelin, 2000 units, weekly.

December 15, 1937: Added vitamin B complex. To continue with liver injections and take one Emmenin tablet daily instead of theelin injections.

February 4, 1938: Stop liver injections. Continue with vitamin B complex and Emmenin tablets, three daily.

March 29, 1938: (Hgb. is now 85%, and R.B.C.'s are normal.) To continue with vitamin B complex and take Emmenin tablets ten days prior to periods. Take thyroid, one-half grain daily.

July 26, 1938: Emmenin discontinued because hemoglobin had dropped to 62%; R.B.C. 3,910,000; W.B.C. 5400. This drop due to excessive flowing which was discovered later to be due to uterine fibroids. A panhysterectomy was done. Blood count returned to normal, Hgb. 85%. From July 26, 1938 to January 5, 1939, her treatment was thyroid, one grain daily; and nicotinic acid, two grains daily.

January 5, 1939: Thyroid discontinued because her basal metabolic rate was now plus 15 per cent. To take capsules of thiamin chloride, 1000 I.U., and nicotinic acid, grains one, night and morning.

April 15, 1939: Resume thyroid. Continue thiamin chloride and nicotinic acid at same dosage.

COMMENTS

The McGill investigators include both nerve and obstructive deafness under the heading of constitutional deafness. It is difficult to imagine that definite degeneration of the auditory nerve is an accompaniment of the run of females suffering from painful, profuse or deficient menstruation.

It is equally difficult to visualize such a tremendous improvement as shown in eight of their cases of females—forty to fifty-five decibels in certain tones—since no attempt is made to theorize regard-

ing the mode of action of estrogens. Oscar Riddle,[22] however, mentions that "Theelin is shown to have a high capacity to raise serum calcium," and elsewhere adds that "The parathyroid hormones apparently mobilize this additional calcium from the bones; however, the parathyroids, directly or indirectly are in turn stimulated to this greater activity by a coincident increase of female sex hormone at this period."

Much can be accomplished to improve the general health and psychology of these unfortunates who have a hearing loss of twenty-five per cent and over by correcting the various nutritional deficiencies and environmental factors. Some improvement in the tone scale can occur through the same means with the addition of estrogenic and other substances.

Albert Gray[1] has stated that he had found extensive degeneration of the nerve with no interference at the stapedial joint in a person who had had very severe deafness—a bit of supportive evidence that otosclerosis may be fundamentally a nerve involvement and that the causes of the conduction interference is secondary and due to other causes, i. e., interference with the nutrition of bone and nerve.

CONCLUSIONS

1. Estrogenic substances probably play an important role in conduction deafness. Estrogen relieves completely in many cases the tinnitus, menstrual irregularities, and other symptoms occurring during menstruation. Unfortunately, these symptoms recur in many instances when estrogen is discontinued.

2. There is definite evidence that thyroid is of importance and also the pituitary and adrenal cortex in treatment.

3. Thiamin chloride and nicotinic acid are exceedingly helpful as may be other factors of the B_2 complex and other vitamins.

4. Neither nerve nor conduction deafness are caused by any single factor but appear to be linked with the various factors related to growth, i. e., endocrine glands, vitamins, mineral salts (the electrolytes) and amino-acids.

5. The evidence, slowly accumulating, points to nutritional deficiencies and the beginnings probably occur during the period of gestation and are due in the main to dietary errors and endocrine disturbances of the pregnant mother. The prevention of deafness, therefore, depends on the mother getting an optimum diet containing all the essential foods during the gestation period, and such

dietary habits must be carried on during babyhood, childhood and adolescence.

6. Finally, further work is necessary concerning the various factors referred to in this and other papers already published, and especially the nutritional, endocrine and allergic factors that may prove to be related to the enlargement of tonsils and adenoids.

384 Post St.

BIBLIOGRAPHY

1. Gray, A.: The Otosclerotic Problem. J. Laryng. & Otol. (Oct.), 1934.

2. Langdon, Brown, Sir Walter: The Endocrines in Theory and Practice. Blakiston, Son & Co., 1937.

3. McLellan, Sir Robert: The Endocrines in Theory and Practice. Blakiston, Son & Co., page 94, 1937.

4. Collip, J., and Campbell, A. D.: Notes on the Clinical Use of Certain Placenta Extracts. Brit. Med. J., 2:1081, 1930.

5. Thompson, W., and Heckel, N.: Sexual Development from an Anterior Pituitary-Like Principle. J. A. M. A., page 1813 (May 28), 1938.

6. Novak, E.: Menopause and Its Management. J. A. M. A., 110:9 (Feb. 26), 1938.

7. Hayden, R. L.: Multiple Specific Nutritional Deficiencies in the Adult. J. A. M. A., 186:261 (Jan. 26), 1936.

8. Mortimer, H., Collip, J., and Wright, R.: Atrophic Rhinitis, the Constitutional Factor and the Treatment with Estrogenic Hormones. Canadian Med. Assn. J., 37:445-446 (Nov. 1), 1937.

9. Burrows, R. B.: Variation Produced in Bones of Growing Rats by Parathyroid Extract. Am. J. of Anatomy, 62:2 (Jan.), 1938.

10. Mellanby, E.: The Experimental Production of Deafness in Young Animals by Diet. J. Physiol., 94:3-380, 1938.

11. Abt, A.: Vitamin C—Pharmacology and Therapeutics. J. A. M. A., pages 1555-1561 (Oct. 22), 1938.

12. Rinehardt, J. F.: Studies Relating to Vitamin C Deficiency. Annals Int. Med., No. 6, 9:671-689 (Dec.), 1935.

13. Ono, T.: Experimental Histopathological Studies of the Ears—A, B, C, Vitaminosis. Trans. Jap. Path. Soc., 18:172-175, 1938.

14. Weber, M., and Becks, H.: The Influence of Diet on the Bone System with Special Reference to the Alveolar Process and Labyrinthine Capsules. J. Am. Dental Assn., 28:196-264 (Feb.), 1931.

15. Gill, H. B., and Stein, T.: Bone Metabolism. J. Bone and Joint Surgery, 18:94-97 (Oct.), 1933.

16. Minot, G. R.: Nutritional Deficiency. Annals Int. Med., 12:4 (Oct.), 1938.

17. Sure, B.: The Little Things in Life. D. Appleton Century Co., 1937.

18. McCarrison, Maj. Gen. Sir Robt.: Nutrition in Health and Disease. Brit. Med. J., pp. 611-614 (Sept. 26), 1936.

19. Spies, T. D.: Personal communication.

20. Mortimer, H.; Wright, H.; Thompson, D., and Collip, J.: Intranasal Administration of Estrogenic Hormones in Constitutional Deafness. Canadian Med. Assn. J., 40:17-21 (Jan.), 1939.

21. Foster, H. C., and Thornton, M. J.: Thyroid in the Treatment of Menstrual Irregularities. Endocrinology, 24:3 (March), 1939.

22. Riddle, O.: The Changing Organism. Cooperation in Research, Carnegie Institution of Washington. Publication No. 501, pp. 259-273, 1938.

IV

RESULTS OF POTASSIUM THERAPY IN NASAL ALLERGY*

HOWARD A. RUSK, M.D.
L. W. DEAN, JR., M.D.
AND
WALLACE RINDSKOPF

ST. LOUIS, MO.

One of us (Rusk)[1-2] has previously reported favorable therapeutic results in chronic uticaria and certain types of bronchial asthma from administration of potassium salts and a diet low in sodium. Bloom[3] and Abt[4] have reported excellent results in various types of nasal allergy with a similar regime.

We have recently studied fifty-five patients with allergic rhinitis. Blood potassium determinations were made in forty-three instances by a new reliable micro method.[5] Table I, which has been previously published,[2] shows our findings in normal individuals and those with urticaria and asthma.

TABLE I

THE POTASSIUM CONTENT OF BLOOD SERUM IN HEALTHY PERSONS AND IN SOME ALLERGIC PATIENTS

NUMBER OF PATIENTS	CONDITION OF SUBJECT	Mg. of Potassium per 100 cc. of Serum		
		LOWEST	HIGHEST	AVERAGE
35	Healthy	17.9	20.6	19.5
20	Urticaria, acute and chronic	22.0	25.3	23.4
10	Bronchial asthma, asymptomatic period	21.3	25.1	23.6
7	Bronchial asthma, acute attack	24.0	25.5	24.4

*From the laboratory of the Jewish Hospital, St. Louis, Missouri, and the Department of Otolaryngology, Washington University School of Medicine, St. Louis, Missouri.

This work was aided by the Edward B. Pryor Memorial Fund.

It may be seen that the potassium content of normal human blood serum is confined within a rather narrow range, i. e., 17.9 and 20.6 mg. per 100 cc. of serum, an average of 19.5 mg. per cent. It should be noted that the accuracy of our method is plus or minus one per cent, so that deviations as minute as one mg. per cent above or below the normal limits represent significant changes.

In the series of patients with allergic rhinitis, presented in this paper, the following classification was made: perennial nasal sensitization, early spring pollinosis, occuring in May and June, and the typical autumnal ragweed hay fever. In certain of these cases determinations were made before and after the onset of symptoms. Table II illustrates our blood potassium studies in these patients.

TABLE II

BLOOD POTASSIUM DETERMINATIONS

	Preseasonal		During Symptoms	
	Average	High	Average	High
Ragweed	18.6	21.3	18.9	20.9
Early spring pollinosis	—	—	19.9	21.7
Perennial	—	—	19.8	21.6

It is obvious from these figures that there is no significant change in the serum potassium in any of our patients with nasal allergy. This is in direct contrast to our findings in urticaria and asthma, and permits the assumption that in allergic rhinitis we are not dealing with a discernible systemic disturbance of the potassium balance as in the instance of asthma and urticaria.

These patients were all advised to reduce materially the amount of sodium in their diet, and were treated with either potassium chloride or potassium gluconate by mouth. The dosage of potassium chloride ranged from 30 to 90 grains per day, given in enteric coated tablets after meals. The potassium gluconate dosage varied from 30 to 120 grains a day and was given in plain tablets with water after meals. In this series of patients there was no local or general manifestation of potassium intolerance or intoxication. All patients continued the drug at least two weeks after the onset of symptoms. In a number of patients potassium chloride in 2.5 per cent and 5 per cent dilutions was used intranasally without appreciable effect. Table III illustrates the results of treatment.

It is well to note that the 1939 hay fever season in St. Louis was particularly severe.

TABLE III

RESULTS OF TREATMENT

Type of Case	No. of Cases	Complete Relief	Marked Relief	Moderate Relief	Slight Relief	No Relief
Ragweed	30	2 (6%)	1 (3%)	4 (14%)	3 (10%)	20 (67%)
Early Spring Pollinosis	15	9 (60%)	5 (33%)	1 (7%)	0	0
Perennial Nasal Allergy	10	2 (20%)	3 (30%)	3 (30%)	1 (10%)	1 (10%)

The results of potassium therapy in the ragweed group were extremely unfavorable, but the three patients who received complete or marked relief are worth considering in detail.

REPORT OF CASES

CASE 1.—Male, age 35; severe ragweed hay fever since boyhood. He was markedly overweight and for four months previous to the ragweed season had been on a low sodium, high potassium, acid ash diet and was taking from 60 to 90 grains of potassium chloride and from three to four grains of thyroid extract daily for the reduction of weight. During this period he lost sixty pounds. He continued the same dosage of potassium during the hay fever season, and for the first time in his memory had absolutely no symptoms of hay fever.

CASE 2.—Male, age 45; severe autumnal hay fever for twenty years. This patient remained entirely symptom-free while on 15 grains of potassium gluconate three times a day. At our request he discontinued the drug for one week and all of his symptoms reappeared. He resumed the potassium and was symptom free in twenty-four hours and continued so throughout the remainder of the season.

CASE 3.—Male, age 30; severe ragweed hay fever for fifteen years. He obtained marked relief from 20 grains of potassium chloride three times a day. He discontinued the drug, and in forty-eight hours all symptoms reappeared. He again obtained prompt relief on the readministration of potassium.

Blood potassium studies, made on one of these patients, showed no deviation from the normal.

Our most striking results were obtained in the mild cases of pollinosis occurring in the spring. All of these patients had typical symptoms and obtained complete or marked relief promptly after taking potassium. This was evidenced by a drying of the nasal mucosa and cessation of rhinorrhea and sneezing. Two of these patients had concomitant asthma, which also cleared up with the alleviation of nasal symptoms. In one of these patients there was a slight elevation (21 mg. per cent) in the blood potassium. The perennial group of cases, due to a variant of allergens, showed improvement in 50 per cent of the cases.

SUMMARY

1. Blood potassium studies in fifty-five patients with nasal allergy disclosed no variation from normal.

2. Of thirty patients suffering from ragweed hay fever and treated with potassium salts, only 9 per cent obtained marked relief. Sixty-seven per cent were not improved.

3. In the treatment of other types of nasal allergy, potassium salts were very effective.

3720 WASHINGTON AVENUE.

BIBLIOGRAPHY

1. Rusk, H. A., and Kenamore, B. D.: Urticaria—A New Therapeutic Approach. Ann. Int. Med., 11:1838 (April), 1938.

2. Rusk, Howard A.; Weichselbaum, T. E., and Somogyi, Michael: Changes in Serum Potassium in Certain Allergic States. J. A. M. A., 112:2395 (June 10), 1939.

3. Bloom, Benson: The Use of Potassium Salts in Hay Fever. J. A. M. A., 111:2281 (Dec. 17), 1938.

4. Abt, Arthur F.: Note on Oral Administration of Potassium Chloride in the Treatment of Hay Fever, Nasal Allergy, Asthma and Sinusitis. Am. Jour. Med. Sc., 198:229 (Aug.), 1939.

5. Weichselbaum, T. E.; Somogyi, Michael, and Rusk, Howard A.: The Determination of Small Amounts of Potassium. J. Biol. Chem. (in press).

V

MÉNIÈRE'S SYMPTOM COMPLEX: A REVIEW OF THE LITERATURE*

KINSEY MACLEOD SIMONTON, M.D.†

ROCHESTER, MINNESOTA

In 1861, Ménière[1-6] described the triad of symptoms, vertigo, tinnitus, and nerve deafness, with remarkable clarity and suggested that the condition was a clinical entity. Since Ménière's report the triad of symptoms has generally been referred to as Ménière's disease. Early writers applied this term, or the term "aural vertigo," to all conditions of the ear of which vertigo was a symptom. Later, those conditions in which the pathologic background could be demonstrated, such as inflammation of the labyrinth or tumors involving the acoustic nerve, were classified under terms of pathologic significance, leaving a group of cases in which the etiologic agent was unknown to be designated by the name of Ménière. For this reason it seems preferable to refer to the triad of symptoms as Ménière's symptom complex, rather than as Ménière's disease.

According to Gowers,[7] Burns described, in 1809, the association of tinnitus aurium and vertigo.

Ménière's report[1] is classic in its description of the attack of vertigo and the intervening symptoms. He noted the sudden onset of vertigo without preceding illness, the recurrence of the attacks, the tendency toward spontaneous remissions of the symptoms, and he emphasized the extreme discomfort of the patient during the attack and the increase of vertigo with movement. He observed that the tinnitus preceded the first attack, persisted between attacks, often increased during the attack and was not affected by pressure on the carotid artery. He noted impaired hearing usually in one, sometimes in both ears, that began with the tinnitus and progressed to complete deafness without history or findings suggestive of disease in the ear.

---

*For the most part based on thesis submitted to the Faculty of the Graduate School of the University of Minnesota, in partial fulfillment of the requirements for the degree of Master of Science in Otolaryngology.

†Section on Otolaryngology and Rhinology, The Mayo Clinic

Ménière[1] reported the case of a young girl who, after traveling all night on the top of a carriage, caught a bad cold that was accompanied by complete deafness and continual vertigo, with vomiting after any motion of the body. The patient died on the fifth day of the illness. At necropsy the only lesion found was a bloody exudate in the semicircular canals. Traces of exudate were found in the vestibule but none was found in the cochlea. The cerebrum, cerebellum and spinal cord were not involved.

Basing his arguments on the experiments of Flourens, Ménière felt justified in relating the symptoms to the pathologic phenomena observed in only one case. He argued against cerebrovascular accident as a cause, because (1) paralysis did not occur, (2) the patient remained conscious, (3) recovery occurred rapidly, (4) attacks were recurrent, (5) neurologic findings were normal and (6) loss of memory was not associated with the attacks. He argued against a relationship of the symptom complex to migraine because of the lack of deafness in the majority of cases of migraine. He considered the labyrinth, and in view of Flourens' work, the semicircular canals especially as the seat of the lesion because (1) injuries to the ear, such as foreign bodies impinging on the ossicles and tearing of the oval window cause symptoms similar to those described, (2) injury of the membrana tympani not involving the ossicles, or suppurative otitis media from colds does not cause complete deafness or vertigo, and (3) sudden inflation when the eustachian tube is obstructed causes momentary giddiness when the membrana tympani is intact but not when the drum has been previously ruptured. Ménière ascribed this to sudden pressure through the ossicles on the oval window when the drum is intact. He quoted experiments on the cerebellum which gave results similar to Flourens' experiments, but since the hearing remained intact in at least one ear, he interpreted this as further evidence in favor of the inner ear. Ménière closed his discussion by admitting that vertigo, nausea and vomiting can come from meningitis, lesions of the cerebrum or of the cerebellum, but he insisted that when accompanied by tinnitus and deafness, the lesion was situated in the inner ear and especially in the semicircular canals.

From the foregoing description it is obvious that Ménière had a clear conception of the symptom complex which we now designate by his name. From the case reported it is equally obvious that this patient did not fulfill the diagnostic criteria of the condition which he had described. In the light of present knowledge, this patient is assumed to have suffered from acute labyrinthitis, and that Mén-

ière linked the clinical findings of his symptom complex with the pathologic entity of acute labyrinthitis.

In subsequent communications,[2-6] Ménière reported nine cases of the typical symptom complex. We must then credit Mèniére with the first comprehensive description of this condition, although, as McKenzie[8] pointed out, his pathologic observations do not explain the clinical phenomena.

Knowledge of the equilibratory function of the labyrinth is based on the work of Flourens[9] who, in 1842, established the fact that the semicircular canals controlled equilibrium and that each canal controlled motion in the plane of that canal. Flourens also noted that there was gradual partial recovery from symptoms due to loss of function of one canal, less from loss of function of more than one canal. Goltz,[10] in 1870, concluded that the vestibular branch of the auditory nerve transmits the impulses that control equilibrium. Ewald[11] noted that the direction of nystagmus was the same as the direction of motion of the endolymph within the semicircular canals. Bárány[12] applied Ewald's experiments to the human being by inducing movement of the endolymph by caloric stimulation or rotation and provided a clinical test of labyrinthine function.

ETIOLOGY AND PATHOLOGY

The etiologic agent and pathologic characteristics of Ménière's symptom complex have not been established. Death seldom occurs during the attack, hence pathologic material is rare. Consequently the literature on the subject contains confusing hypotheses interspersed with a few serious experimental attempts to establish facts. This situation led Christie[13] to state that "At the present this name (Ménière's disease) is used for any collection of aural symptoms for which the user does not know some other name."

Ménière[1] felt that he had found the cause of the condition when he observed at postmortem examination the gross effects of inflammation in the labyrinth. Knapp,[14] in 1871, listed eleven causes of vertigo: (1) a group of unknown etiology, (2) trauma, (3) hemorrhage into the labyrinth, (4) meningitis, (5) chronic suppurative otitis media, (6) childbirth, (7) eclampsia infantum, (8) acute exanthemata, (9) progressive deafness after childbirth, (10) sunstroke and (11) syphilis, and he stated that "If their inflammatory nature is not always established by sufficient pathologic evidence, they may at least be supposed to have their origin in a structural change in the labyrinth." Guye,[15] in 1880, wrote that "Most cases

are secondary to inflammation of the tympanum or antrum." He included under the term "Ménière's disease," all cases of vertigo with aural symptoms, and reported four cases in which vertigo was caused by inflammation of the ear.

Gottstein[16] reported as Ménière's disease a series of cases, apparently of epidemic meningitis in which absolute bilateral deafness developed, and in which after recovery, staggering in the dark occurred; he also reported two cases of leukemia associated with sudden deafness and dizziness, and one case of tabes associated with sudden deafness.

Lake,[17] in 1912, classified aural vertigo according to cause in the following manner:

1. Peripheral causes.
   (a) Chronic progressive middle ear deafness (in young persons).
   (b) Labyrinthine hemorrhage and embolism (in old persons).
   (c) Traumatic aural vertigo caused by basal skull fractures, impactions of cerumen, excessive nose blowing (action on round window).

2. Vertigo due to altered blood pressure.
   (a) Increased blood pressure, arteriosclerosis and so forth.
   (b) Diminished blood pressure.

3. Aural vertigo associated with general systemic causes such as:
   (a) Leukemia.
   (b) Occasional, such as gout.
   (c) Ocular symptoms (visual aura).
   (d) Syphilis.
   (e) Cerebral anemia.

Disturbance of function of the eustachian tubes has been said by Scott[18] to be the cause of the symptom complex. In 1920, he wrote that "The cause, often so subtle to find, and consequently so often overlooked, though admitted as the cause when recognized, is,

in my experience, simply unilateral eustachian insufficiency." Shield,[19] in 1936, stated that in cases of myxedema, pseudo-Ménière's symptoms (vertigo without deafness) are due to occlusion of the eustachian tube and disturbed function of the middle ear from swollen mucous membranes.

Ménière's symptom complex has been reported as caused by encephalitis, by Videbech;[20] by acquired syphilis, by Gross,[21] and by inhalation of benzine gas, by Ruttin.[22] Hofer[23] reported a case in which Ménière's triad of symptoms was exhibited for three weeks prior to death. At necropsy, he found thrombosis of the basal cerebral artery with infarction that involved the fourth ventricle. This is suggestive of the possibility of central origin of the symptoms. Shield[19] credited Brunner with the statement that in 42 per cent of cases of concussion of the brain, injury to the vestibular and cochlear nuclei occurred, and these cases were referred to as "concussion of the brain with ear symptoms." These patients suffer mild vertigo in attacks.

Just[24] believed that the symptoms are secondary to infection of the ethmoid and sphenoid sinuses and stated that the condition is relieved by treating the sinusitis. Shambaugh[25] stated his belief that diplacusis associated with Ménière's symptom complex indicates a state of inflammation of the labyrinth secondary to a focus of infection, and he reported two cases in which relief was obtained by removal of foci of infection.

Fraser[26] reviewed the literature in regard to patients who had leukemia who exhibited Ménière's symptom complex and concluded that in cases of disease characterized by bleeding, there is occasional hemorrhage into the inner ear. If death follows soon, blood or exudate are found; if much later, bony and fibrous changes are present in the labyrinth. Fraser credited Alexander with the claim that Ménière's patient had suffered from leukemia.

Thornval[27] stated that in most cases of Ménière's disease otolithic symptoms are present; that during the attack they must hold the head in a characteristic position. He found that in the attack the direction of nystagmus can be reversed by caloric stimulation. He suggested that the macula utriculi, and not the semicircular canals, may be the seat of the disturbance. Tumarkin[28] stated that "Ménière's syndrome is referable to some degeneration, whether toxic, inflammatory, or abiotrophic of the inner ear. Whether the lesion is primarily in the neuro-epithelium of the inner ear, or * * * in the eighth nerve itself is not definitely proven." Werner,[29] in

experiments on fishes, found that destruction of the macula utriculi produced the same changes as loss of the entire labyrinth. He did not discover the function of the macula sacculi.

Knapp,[14] and later Cheatle,[30] compared Ménière's symptom complex to glaucoma of the eye, Cheatle suggesting that the condition may be caused by increased pressure in the labyrinth, either by increased production or impaired drainage of the perilymph. Portmann[31] believed that the symptoms were due to an increase of pressure produced through the saccus endolymphaticus and credited Guild with showing that the saccus endolymphaticus is the route of outflow of the endolymph. Aboulker[32] cited chronic hypertensive meningitis as the cause.

Reflex disturbance of circulation along the vertebral artery and its branches has been considered as the cause of Ménière's symptom complex by Woakes,[33] Bozzi[34] and Koch.[35] Mogan and Baumgartner[36] reported a case in which symptoms were relieved after bilateral cervical sympathectomy.

Mygind and Dederding[37] and Dederding[38] observed an increase in body fluids of patients who had Ménière's disease. Of their patients, 60 per cent were overweight, 20 per cent were underweight, and they noted an increase in symptoms with increase of weight, improvement with loss of weight. In summary, they stated: "Ménière's is not a disease sui generis, but a typical reaction of a predisposed labyrinth to an almost infinite series of exo- and endogenic influences, which, however, have this in common, that they express themselves through the vessels, especially the capillaries." They believed this to be extracellular edema due to deficient function of capillaries; nonpitting because of adequate lymph drainage.

Dohlman[39] cited experiments by Skoog, who produced symptoms similar to those of Ménière's symptom complex in guinea pigs by injecting into one of the vertebral arteries an antigen that produced anaphylaxis. There was no such response on injection into the carotid artery or into a vein, or if India ink was first injected into the artery. The reaction occurred in animals that previously had bilateral labyrinthectomy. Dohlman suggested that a localized central region is responsible for Ménière's symptom complex.

Dandy,[40] in 1937, concluded that "only a lesion in the sensory root of the vestibular branch can cause Ménière's disease." Dandy quoted Wittmaak who, in 1930, reported two patients who had Ménière's symptom complex with concretions in the ductus coch-

learis and one who had a minute neuroma in the cochlea; he also quoted Just who had included a reference by Zange. Zange had found a small hematoma in the brain stem near one of the nuclei of the auditory nerve. Dandy[41] also suggested that "By analogy with other conditions it is easier to believe that the nerve itself and not the end-organ may be primarily involved"; the attacks are an explosion of hyperfunction of the inner ear due to a lesion on the nerve. He expressed his belief that the lesion is situated on the nerve, because both hearing and equilibrium are affected, and that a lesion affecting both end-organs would be too diffuse not to be found. He later suggested that a lesion was situated in the central pathways.[42] Crowe,[43] in reviewing Dandy's cases, suggested that the lesion is either on the nerve where the two branches pass through a common sheath, or in the endolymph, and that the lesion is both irritative and degenerative in nature.

Munro,[44] in 1937, reviewed the recent literature and found a variety of causes given for Ménière's symptom complex. These are:

(1) An explosion of overfunction of the labyrinth (Thornval).

(2) Lead poisoning (Whitworth).

(3) Sensitivity to orris root (Yandell).

(4) Toxicity from any cause, such as tobacco (Brain).

(5) A temporary accumulation of fluid in the labyrinth, "of such nature as to class this disease with epilepsy, eclampsia, migraine, angina pectoris, bronchial asthma, allergic disease, gout, et cetera" (Foldes).

(6) Focal infection (McMurray).

(7) Sensitivity to orris (Malone).

(8) Overeating of meat (Muck).

(9) Tumors of pons and cerebellopontine angle.

(10) Arachnoiditis.

(11) Syphilitic meningitis.

(12) Aneurysm of the basilar artery.

(13) Pressure on the auditory nerve by normal or abnormal vessels.

SYMPTOMS

Ménière's symptom complex is comprised of the triad of symptoms, vertigo, nerve deafness and tinnitus. Nausea and vomiting are very often present during the attack. For purposes of distinguishing it from other conditions presenting some or all of the symptoms of the triad, detailed attention to the symptoms presented by the patient is essential. Due to the lack of objective findings other than impaired hearing, one must depend entirely on the history in arriving at the diagnosis. There have been attempts in the literature to describe the symptoms accurately, especially with reference to aids in lateralization of the lesion in patients destined for surgical treatment, but frequently such attempts have been contradictory.

Ménière[1] in his original paper gave a classic description of the symptoms, to which little has been added since then. Féré and Demars,[45] in 1881, presented a detailed account of the symptomatology together with several complete histories of cases.

Vertigo during the paroxysms is the most striking feature of Ménière's symptom complex. Ménière stressed the sudden onset, disabling severity and complete recovery from the vertigo. He noted the recurrence of severe prostration at the slightest motion and also the tendency toward giddiness which persists between attacks. Dandy[46] more recently pointed out the periodic recurrence of sudden, severe vertigo, as distinguished from the constant mild vertigo experienced by patients who have lesions of the cerebellopontine angle.

The sensation of rotation was described by Gowers[47] as one of the characteristics of this vertigo. The rotation is usually in a horizontal plane to right or left, although it may be in the vertical plane, either forward or backward.

The severity of the vertigo may cause the patient to fall during the attack. Féré and Demars[45] stated that the patient may fall away from or toward the affected side, but that the same patient always falls in the same direction. Curschmann[48] stated that the patient falls toward the side affected. The same confusion exists concerning the direction of the nystagmus. Curschmann said that the nystagmus is toward the side affected. McKenzie[49] observed nystagmus toward the side affected in one case; in another it was in both directions in a horizontal plane as well as upward in a single attack. Jackson[50] noted both nystagmus and sensation of objects rotating to the right in a case of left sided deafness.

Mild to severe nausea and sudden projectile vomiting frequently accompany the attacks. Ménière[1] noted that vomiting has no rela-

tion to the state of fullness or emptiness of the stomach. After the stomach has been emptied retching may continue.

Deafness commonly involves the nerve. It is gradually progressive and tends to increase during the attack. Charcot[51] stated that when the deafness is complete the vertigo ceases. Deafness may be bilateral but usually is more severe on the side of the involved ear. Dandy[46] and Geise[52] reported cases of typical vertigo but without deafness.

Tinnitus, like the deafness, is increased during the attack and may disappear between attacks. The early literature contains detailed descriptions of the tinnitus. It is most frequently described as high pitched and whistling.

Headache frequently accompanies Ménière's symptom complex. Cohen[53] reviewed 126 cases and found headache present in 114 (91 per cent). Of his own eleven patients, five had definite headache, two, pain in the head. The headaches were not characteristic in type or in occurrence Dederding[38] reported headache in 91 per cent. Collet,[54] in 1937, described a sense of numbness in the ear and corresponding cheek which accompanied intense vertigo in Ménière's symptom complex, and reported four cases in which this phenomenon occurred. He ascribed this finding to "angiospasm of the vestibular centers or pathways which extends into neighboring territory, the nuclei of origin of the trigeminus nerve." Keen[55] stated that chronic pharyngitis frequently accompanies Ménière's symptom complex. Berggren[56] observed a positive Babinski sign during and immediately after the attack in four cases. He inferred that there is a transient functional disturbance in the pyramidal tract during the attack.

TREATMENT

The treatment of Ménière's symptom complex has been more varied than the hypotheses regarding its etiologic and pathologic characteristics. Due to the tendency to spontaneous remissions, almost every form of treatment advocated has given promise of success. The lack of general acceptance testifies to the failure of the majority of these measures.

Ramadier,[57] in 1933, reviewed the treatment and made the following classification:

1. Medical treatment.
   (1) Sedative or symptomatic.
      (a) Suppression of all causes of vestibular irritation.
      (b) Physical, ice cap and so forth.
      (c) Medical, hypnotics and so forth.

(2) Based on etiology and pathology.

(a) Drugs.

1. Adrenalin.
2. Atropine.
3. Pilocarpine.
4. Nitrates.
5. Acetyl choline.
6. Quinine (Charcot).

(b) Dietetic.

1. Liquid diet one day each week.
2. Dehydration (Dederding).

(c) Physiotherapeutic.

1. High frequency current.
2. Diathermy.
3. Ionization of potassium iodide.
4. Radiotherapy.

2. Surgical therapy.

(1) Conservative.

(a) Lumbar puncture (Babinski, 1904).
(b) Decompression trephine of the cerebellar fossa (Aboulker).
(c) Retropetrous trephine decompression puncture of the pontocerebellar lake.
(d) Puncture of the endolymphatic duct (Portmann).
(e) Occipital trephine decompression (Cushing).
(f) Decompression of the inner ear (membranous labyrinth).

(2) Radical operation.

(a) Destructive trephination of the inner ear.
(b) Section of the auditory nerve.

Suggestions for treatment noted in the literature are: Iodides (Shield[19]), epinephrine by mouth in doses of 20 to 30 minims twice daily (Barbazon[58]), quinine in doses of 0.6 to 1.0 gm. daily until the tinnitus is changed to that of quininism (Charcot[59]), removal

of cerumen (Jackson[60]), eustachian tube inflations (Scott[18]). Dehydration, by means of purgation, hot packs, baths and sweats has long been used with some benefit; notably relief of the immediate attack.

Mygind and Dederding[37] and Dederding[38] observed an increase in body fluids in cases of Ménière's symptom complex. They believed that this was due to extracellular edema secondary to deficient function of capillaries, and suggested that retention of water in the ears produces the aural symptoms. The treatment they proposed consists of restricted intake of fluids (700 cc. daily), a diet low in content of salt to promote diuresis, a reduction diet for patients who are overweight; exercise, massage and light therapy to stimulate vasomotor tone. They report that after three years sixty-seven of eighty-three patients did not have recurrence of attacks and all but eight patients had improved hearing. In association with Mygind and Dederding, Neilsen[61] studied water metabolism and found high retention of fluid in cases of Ménière's symptom complex. After treatment, patients showed less retention of fluid, which led to the deduction that the disturbed water metabolism is corrected during the low fluid, low salt regimen.

Furstenberg, Lashmet and Lathrop[62] assumed that when water accumulates in the body, minerals also accumulate. In a series of controlled experiments they found that (1) retention of water produced the attack, (2) retention of water without sodium did not produce the attack, (3) loss of water and retention of sodium produced the attack and (4) loss of water and sodium did not produce the attack. From these findings they assumed that it is retention of the sodium ion and not of the water that induces the attack and proposed a regimen of a low intake of sodium with substitution of ammonium chloride. They reported treatment of 125 patients with "splendid" results. These patients were treated in the hospital and care was taken to eliminate all sodium from the diet.

Brown[63] reported twelve patients relieved of vertigo by the Furstenberg regimen. Patients who suffered from vertigo without deafness were not relieved.

Physical therapy has been advocated for the treatment of Ménière's symptom complex. Russell,[64] in 1929, reported a patient treated by diathermy whose condition improved but later a relapse occurred. Keen[55] treated twenty patients by the electrophonoid vibromassage of Zund-Burguet. Of eight patients reported, all were relieved of dizziness, tinnitus was relieved in some; the hearing remained unchanged.

Surgical treatment has had many advocates and many operations have been proposed. Charcot[59] suggested cautery to the skin over the mastoid and Parry[65] recommended insertion of a Seton drain into the skin over the mastoid process for a period of six months.

More rational surgical approaches to the problem range from cerebrospinal drainage as advocated by Babinski and Putnam,[66] in 1908, to the currently favored section of the vestibular nerve. The other surgical measures employed for relief of vertigo have been in general, decompression of the cerebellar fossa or the endolymph spaces, and destructive operations on the labyrinth.

Aboulker[32] noted improvement in vertigo following lumbar puncture, and was led to perform trephine decompression operations behind the mastoid. In 1927, he reported two cases of five-year cures following this procedure, both with and without opening the dura. He advised that "the decompression be done . . . in every case in which the classic treatments fail, whether or not the manometric puncture (spinal) shows hypertension."

Portmann[31] believed that the symptoms were due to endolabyrinthine hyperpressure and advocated opening and draining the saccus endolymphaticus. Parkinson,[67] in 1935, reported a case in which operation was performed after Portmann's method with relief of symptoms.

McNally,[68] in 1926, published results of experiments on the saccus endolymphaticus in rabbits, which refute Portmann's hypothesis of endolabyrinthine hypertension. In summary he stated: "The rabbit shows very little disturbance of its vestibular mechanism following incision, or cauterization, or the application of pressure to one saccus endolymphaticus." McNally noted that the labyrinthine reflexes were not interfered with by total ablation of the saccus.

The British surgeons, following the teaching of Cheatle,[30] that Ménière's symptom complex was caused by increased pressure in the labyrinth, and his suggestion in 1897, that such patients might be relieved by decompression of the labyrinth, have resorted to various operations on the labyrinth and have reported relief of symptoms in most cases.

Lake[17, 69-71] opened the lateral semicircular canal and the vestibule. In later cases he removed the stapes and opened the oval window as well. In 1911 he reported relief from vertigo of ten patients treated in this manner. All were totally deaf on the affected sides after operation, indicating destruction of the labyrinth. Jenkins[72] made a small opening into the lateral canal with relief of ver-

tigo and without change in hearing. Kelson[73] "uncapped" the lateral canal with recovery. Mollison[74, 75] reported opening the lateral canal and in later cases also the injection of absolute alcohol into the opened canal with relief of vertigo in 73 per cent of patients. Quix[76] opened the lateral canal only. Milligan[77, 78] performed a "bridge" operation, opening both the lateral canal and the vestibule. In twenty cases he reported no fatalities, 100 per cent relief of vertigo, 40 per cent complete, and 20 per cent partial relief of tinnitus, and complete loss of hearing. Davis[79] did vestibulotomy only. Jones[80] reported three cases. In the first, he opened and curetted the lateral canal and ampulla without relief. Syphilis was suspected as the causal agent in this case. In the second, he destroyed all three canals with relief of symptoms; in the third, he destroyed the lateral canal and vestibule with complete loss of hearing. Thursfield[81] performed a complete labyrinthectomy with partial relief of vertigo, and Yorke[82] reported destroying the labyrinth with relief of vertigo, diminution of tinnitus, and complete loss of hearing.

American neurologic surgeons have shown a preference for intracranial division of the auditory nerve in the treatment of persistent recurring vertigo such as Ménière's symptom complex. Frazier,[83] in 1912, reported division of the nerve in a case of complete deafness and persistent vertigo following influenza. The patient was only partially relieved of vertigo. In 1913[84] he reported a similar operation on a patient who had persistent tinnitus after a traumatic basal skull fracture. The tinnitus was relieved.

Dandy,[40, 85] referring back to Charcot who, in 1874, suggested intracranial division of the auditory nerve for the relief of Ménière's symptom complex, again proposed this means of treatment. In his earlier cases, Dandy[85] divided the entire nerve, with relief of vertigo but total deafness resulted. In 1933, he[86] reported partial division of the nerve with relief of vertigo but without significant change in hearing. Later[87] he reported that in performing hemisection of the auditory nerve, some of the cochlear fibers are sectioned, yet there is no significant change in the audiogram except for the highest tones; he referred to work on the fifth nerve in cases of tic douloureux[88] in which partial section did not cause significant change in sensation and to hemisection of the optic nerve[89] with subsequent good vision. In 1934,[90] he reported relief of vertigo from unilateral hemisection of the auditory nerve in cases with bilateral deafness and tinnitus. In these cases the side on which deafness was greater was selected for operation. From this observation he questioned whether there is bilateral involvement.[90] After an experience with

two cases in which nine-tenths of the auditory nerve was divided without significant loss of hearing,[91] Dandy performed a bilateral hemisection of the anterior five-eighths of the auditory nerves of a patient who had tinnitus and vertigo but who retained normal hearing and whose condition was diagnosed as "pseudo Ménière's disease."[42] The results of bilateral section of the vestibular nerves were (1) relief of vertigo, (2) absent responses to caloric and whirling tests, (3) staggering in the dark and (4) inability to focus the eyes sharply.[92]

Crowe,[43] in 1938, reviewed the results of vestibular nerve section experienced by ninety-four patients operated on by Dandy. Of these, the entire auditory nerve was divided in forty-nine cases and partial division was done in forty-five. There was complete relief of vertigo in all cases, tinnitus was improved in some and was unchanged or made worse in others. Of those patients who had partial nerve section, hearing was reported as improved in 22 per cent; in others, the hearing grew progressively worse.

Coleman and Lyerly,[93] in 1933, reported ten cases in which treatment consisted of division of the auditory nerve. Relief of vertigo was complete in all cases, although one patient retained vestibular responses after the operation. This finding should indicate that the division of the vestibular branch was incomplete. Swift, in 1933,[94] reported one patient operated on after the method of Dandy with relief, and Rutherford, in 1937,[95] reported a patient who died of parotitis after operation. Rutherford did not report pathologic studies on his patient.

Since the present writing, Walsh and Adson[96] have reviewed 182 cases of Ménière's symptom complex in which treatment was given at The Mayo Clinic. They concluded that: (1) Spontaneous recovery is doubtful, although remissions of symptoms frequently occur. (2) Complete deafness or loss of vestibular reactions on the affected side is no guarantee for disappearance of vertigo. (3) Medical treatment by a modification of the Furstenberg regimen is of definite value. In their series, 34 per cent were completely relieved of vertigo, 28 per cent were partially relieved and 38 per cent experienced no relief. Results were more favorable among patients who had unilateral deafness and tinnitus than among those who had bilateral deafness. Hearing was reported improved in 28 per cent and tinnitus, in 33 per cent. Thirty-five patients were completely relieved of vertigo for periods of six months to three years after treatment was discontinued. (4) Total or subtotal intracranial section of the acoustic

nerve is the treatment of choice for patients not relieved by medical management.

THE MAYO CLINIC.

REFERENCES

1. Ménière, P.: Mémoire sur des lésions de l'oreille interne donnant lieu à des symptômes de congestion cérébrale apoplectiforme. Gaz. méd., Paris, 3 s., 16:597-601, 1861.

2. Ménière, P.: Sur une forme particulière de surdité grave dépendant d'une lésion de l'oreille interne. Gaz. méd., Paris, 3 s., 16:29, 1861.

3. Ménière, P.: Sur une forme de surdité grave dépendant d'une lésion de l'oreille interne. Bull. Acad. de méd., Paris, 26:241, 1860-1861.

4. Ménière, P.: Maladie de l'oreille interne offrant les symptômes de la congestion cérébrale apoplectiforme. Gaz. méd., Paris, 3 s., 16:88, 1861.

5. Ménière, P.: Nouveaux documents relatifs aux lésions de l'oreille interne caractérisées par des symptômes de congestion cérébrale apoplectiforme. Gaz. méd., Paris, 3 s., 16:239, 1861.

6. Ménière, P.: Observations de maladies de l'oreille interne caractérisées par des symptômes de congestion cérébrale apoplectiforme. Gaz. méd., Paris, 3 s., 16:379-381, 1861.

7. Gowers, W. R.: Diseases of the Nervous System. P. Blakiston, Son & Co., Philadelphia, pp. 1123-1131, 1888.

8. McKenzie, Dan: Ménière's Original Case. J. Laryng. & Otol., 39:446-449 (Aug.), 1924.

9. Flourens, P.: Du système nerveux dans les animaux vertébrés. J. B. Baillière, Paris, p. 516, 1842.

10. Goltz: Quoted by Knapp, Hermann.

11. Ewald: Quoted by Kerrison, P. D.: Diseases of the Ear. J. B. Lippincott Company, Philadelphia, fourth edition, pp. 279-291, 1930.

12. Bárány: Quoted by Kerrison, P. D.: Diseases of the Ear. J. B. Lippincott Company, Philadelphia, fourth edition, pp. 279-291, 1930.

13. Christie, J. F.: Neuro-otology: Its Relation to General Medicine and Surgery. Glasgow M. J., 124:73 (July), 1935.

14. Knapp, Hermann: A Clinical Analysis of the Inflammatory Affections of the Inner Ear. Arch. Ophth., 2:204-283, 1871.

15. Guye, A.: On Ménière's Disease; Conclusion. Arch. Otolaryng., 9:230-240, 1880.

16. Gottstein, J.: On Ménière's Complex of Symptoms. Arch. Otolaryng., 9:255-268, 1880.

17. Lake, R.: Aural Vertigo (Non-Suppurative): A Clinical and Therapeutical Study. Lancet., 2:1638-1642 (Dec. 14), 1912.

18. Scott, Sydney: Vertigo, Especially in Respect of Its Surgical and Medical Treatments. Lancet., 1:535-537 (Mar. 6), 1920.

19. Shield, J. A.: Discussion of Ménière's Disease in Head Injuries and Myxedema. South. M. J., 29:193-197 (Feb.), 1936.

20. Videbech, H.: Affection du labyrinthe se présentant sous les traits du syndrome de Ménière (exploration fonctionelle et examen microscopique du labyrinthe). Acta oto-laryng., 22:51-65, 1935.

21. Gross, J. C.: Síndrome de Ménière bilateral, con restitutio ad integrum del lado derecho. Sordera de la percepción del lado izquierdo. Ligera hiperexcitabilidad a la prueba calórica mínima. Excitabilidad normal a la prueba calórica máxima y rotatoria. Ausencia clínica de signos de sífilis. Rev. de med. y cir. de la Habana, 37:532-535 (July 31), 1932.

22. Ruttin, Erich: Ohrbefunde bei Benzindampfintoxication. Acta oto-laryng., 23:410-413, 1936.

23. Hofer, I.: Ménière's Symptom Complex After Thrombosis of the Basal Cerebral Artery. Abstr. in: Arch. Otolaryng., 21:99-100, 1935.

24. Just, H.: Etiology and Therapy of Ménière's Disease. Abstr. in: Arch. Otolaryng., 20:586 (Oct.), 1934.

25. Shambaugh, G. E., Jr.: Significance of Diplacusis in Ménière's Syndrome. Medical Papers, Christian Birthday Volume, pp. 894-902, 1936.

26. Fraser, J. S.: Affections of the Labyrinth and Eighth Nerve in Leukemia. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 37:361-371 (Mar.), 1928.

27. Thornval: Quelques considérations sur la pathogénie de la maladie de Ménière. Acta oto-laryng., 13:4-22, 1928.

28. Tumarkin, I. A.: Otolithic Catastrophe: A New Syndrome. Brit. M. J., 2:175-177 (July 25), 1936.

29. Werner, C. F.: The Static Function of the Macula Utriculi and its Otoliths. Abstr. in: J. Laryng. & Otol., 51:815-816 (Dec.), 1936.

30. Cheatle, A. H.: The Conducting Portion of the Labyrinth. Arch. Otolaryng., 26:185-187, 1897.

31. Portmann, Georges: The Saccus Endolymphaticus and an operation for Draining the Same for the Relief of Vertigo. J. Laryng. & Otol., 42:809-817 (Dec.), 1927.

32. Aboulker, Henri: Pathogénie et traitement chirurgical du vertige de Ménière. Presse méd., 2:1412-1413 (Nov. 19), 1927.

33. Woakes, Edward: On Deafness, Giddiness and Noises in the Head. Am. J. M. Sc., 127:210-213, 1879.

34. Bozzi, E.: Internal Auditory Artery in Relation to Arteriosclerosis. Abstr. in: Arch. Otolaryng., 20:583 (Oct.), 1934.

35. Koch, J.: Ménière's Disease and Mechanical and Psychic Trauma. Abstr. in: Arch. Otolaryng., 17:108, 1933.

36. Mogan, R. F., and Baumgartner, C. J.: Ménière's Disease Complicated by Recurrent Interstitial Keratitis; Excellent Result Following Cervical Ganglionectomy; Report of Case. West. J. Surg., 42:628-631 (Nov.), 1934.

37. Mygind, S. H., and Dederding, D.: Significance of Water Metabolism in General Pathology as Demonstrated by Experiments on the Ear. Acta oto-laryng., 17:424-466, 1932.

38. Dederding, D.: Our Ménière Treatment (Our Principles and Results). Acta oto-laryng., 16:404-415, 1931.

39. Dohlman, Gösta: Some Practical and Theoretical Points in Labyrinthology. Abstr. in: J. Laryng. & Otol., 50:779-790, 1935.

40. Dandy, W. E.: Pathologic Changes in Ménière's Disease. J. A. M. A., 108:931-937 (Mar. 20), 1937.

41. Dandy, W. E.: Ménière's Disease: Diagnosis and Treatment; Report of Thirty Cases. Am. J. Surg., 20:693-698, 1933.

42. Dandy, W. E.: Treatment of So-called Pseudo-Ménière's Disease. Bull. Johns Hopkins Hosp., 55:232-239 (Sept.), 1934.

43. Crowe, S. J.: Ménière's Disease: A Study Based on Examinations Before and After Intracranial Division of the Vestibular Nerve. Medicine, 17:1-36 (Feb.), 1938.

44. Munro, Donald: The Surgical Treatment of Certain Repeated Explosions of Vertigo Occurring in the Absence of any Demonstrable Etiology—Ménière's Disease. New England J. Med., 216:539-551 (Apr. 1), 1937.

45. Féré, Ch., and Demars, Achille: Note sur la maladie de Ménière et en particulier sur son traitement par la méthode de M. Charcot. Rev. de méd., Paris, 1:796-820, 1881.

46. Dandy, W. E.: Ménière's Disease: Diagnosis and Treatment. Tr. Am. Otol. Soc., 24:120-125, 1934.

47. Gowers, W. R.: The Diagnosis of Auditory Vertigo. Brit. M. J., 2:274-275 (Aug. 26), 1876.

48. Curschmann, Hans: Textbook on Nervous Diseases. P. Blakiston's Son & Co., Philadelphia, 2:839-840, 1915.

49. McKenzie, Dan: Nystagmus During the Ménière Attack. J. Laryng. & Otol., 39:322-323 (June), 1924.

50. Jackson, Hughlings: Auditory Vertigo. Brain, 2:29-38, 1879.

51. Charcot, M.: Quoted by Féré, Ch., and Demars, Achille.

52. Geise, A.: Ménière's Disease Without Involvement of the Cochlear Nerve. Abstr. in: Arch. Otolaryng., 11:224-225, 1930.

53. Cohen, L. H.: Headache as a Feature of Ménière's Symptom Complex. Guy's Hosp. Rep., 85:215-224 (Apr.), 1935.

54. Collet, F. J.: Phenomenon of the "Dead Cheek" Accompanying the Vertigo of Ménière. Abstr. in: Arch. Otolaryng., 25:479, 1937.

55. Keen, J. A.: The Treatment of the Ménière Syndrome by the Zünd-Burguet Electrophonoid Vibromassage. Lancet., 1:285-286 (Feb. 6), 1932.

56. Berggren, S.: A Positive Babinski Sign in Ménière's Syndrome. Abstr. in: Arch. Otolaryng., 18:535, 1933.

57. Ramadier, J.: Le traitement du vertige. Ann. d'oto-laryng., pp. 51-71 (Jan.), 1933.

58. Barbazon: The Treatment of Vertigo by Epinephrin. Abstr. in: Arch. Otolaryng., 1:113, 1925.

59. Charcot, J. M.: Lectures on the Diseases of the Nervous System, Second Series. London, New Sydenham Society, pp. 261-274, 1881.

60. Jackson, Hughlings: Remarks on a Case of Vertigo, Reeling and Vomiting From Ear Disease. Lancet., 2:334-335, 1873.

61. Neilsen, S. F.: Water Tests in Otology; Experiments with Water Charging. Acta oto-laryng., 16:415-429, 1931.

62. Furstenberg, A. C., Lashmet, F. H., and Lathrop, Frank: Ménière's Symptom Complex: Medical Treatment. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 43:1035-1046 (Dec.), 1934.

63. Brown, Madelaine R.: The Medical Treatment of Ménière's Syndrome. J. A. M. A., 108:1158-1160 (Apr. 3), 1937.

64. Russell, E. K.: Case of Ménière's Disease Treated by Diathermy. Brit. J. Actinotherapy, 3:250-251 (Mar.), 1929.

65. Perry, T. W.: On the Differential Diagnosis Between Ménière's Disease and Other Cases Exhibiting Ménière's Complex of Symptoms; with Remarks on the Practical Value of the Seton in Obstinate Cases of Both Conditions, Together with Case Illustrating Excellent Results Obtained by Seton in the Latter Condition. Brit. M. J., 1:1107-1110 (May 11), 1907.

66. Babinski and Putnam: (Quoted by Starr, M. A): Organic and Functional Nervous Diseases: A Textbook of Neurology. Lea and Febiger, New York, P. 898, 1913.

67. Parkinson, S. N.: Ménière's Symptom Complex: Endolymph Decompression with Symptomatic Improvement; Report of Case. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 44:382-386 (June), 1935.

68. McNally, W. J.: Experiments on the Saccus Endolymphaticus in the Rabbit. J. Laryng. & Otol., 41:349-360 (June), 1926.

69. Lake, Richard: Removal of the Semicircular Canals in a Case of Unilateral Aural Vertigo. Lancet., 1:1567-1568 (June 4), 1904.

70. Lake, Richard: A Case of Operation on the Vestibule for the Relief of Vertigo; Together with a Description of the Flap Employed in Order to Obtain a Better View of the Parts During Operation; with Remarks on the History of the Operation. Lancet., 1:26-28 (Jan. 6), 1906.

71. Lake, Richard: Ten Cases of Operation for Ménière's Disease (Aural Vertigo). Lancet., 1:1569-1570 (June 10), 1911.

72. Jenkins, G. J.: Labyrinthine Vertigo (Ménière's Symptoms-Non-Infective) Treated by Operation. Proc. Roy. Soc. Med. (Sect. Otol.), 4:116-120, 1911.

73. Kelson, W. H.: Operation for Ménière's Symptoms. J. Laryng. & Otol., 30:352-353 (Sept.), 1915.

74. Mollison, W. M.: Case of Vertigo Cured by Opening the External Semicircular Canal. J. Laryng. & Otol., 38:539-541, 1923.

75. Mollison, W. M.: The Operative Treatment of Vertigo. J. Laryng. & Otol., 51:38-42 (Jan.), 1936.

76. Quix, F. H.: Quelques points importants dans le pronostic et le traitement des labyrinthites. Ann. d'oto-laryng., pp. 837-842 (Aug.), 1935.

77. Milligan, W.: Severe Labyrinthine Vertigo (Ménière's Disease): Operation, Recovery. Proc. Roy. Soc. Med. (Sect. Otol.), 5:73 (Feb. 16), 1912.

78. Milligan, William: The Surgical Treatment of Suppurative and Certain Nonsuppurative Affections of the Labyrinth. J. Laryng. & Otol., 39:245-252 (May), 1924.

79. Davis, E. D. D.: Severe Aural Vertigo Treated by Superior and Inferior Vestibulotomy. J. Laryng. & Otol., 43:734-735, 1928.

80. Jones, H. E.: Three Cases of Operation on the Labyrinth for Vertigo (Nonsuppurative). Proc. Roy. Soc. Med. (Sect. Otol.), 5:75-79, 1911-1912.

81. Thursfield, Hugh: Cerebral Tumor. Proc. Roy. Soc. Med. (Sect. Neurol.), 9:56 (Feb. 24), 1916.

82. Yorke, C.: Ablation of the Labyrinth in a Case with Ménière's Symptoms. Brit. M. J., 2:429, 1918.

83. Frazier, C. H.: Intracranial Division of the Auditory Nerve for Persistent Aural Vertigo. Surg., Gynec. & Obst., 15:525-529 (Nov.), 1912.

84. Frazier, C. H.: Intracranial Division of the Auditory Nerve for Persistent Tinnitus. J. A. M. A., 61:327-329 (Aug. 2), 1913.

85. Dandy, W. E.: Ménière's Disease: Its Diagnosis and a Method of Treatment. Arch. Surg., 16:1127-1152 (June), 1928.

86. Dandy, W. E.: Treatment of Ménière's Disease by Section of Only the Vestibular Portion of the Acoustic Nerve. Bull. Johns Hopkins Hosp., 53:52-55 (July), 1933.

87. Dandy, W. E.: Effect of Hemisection of Cochlear Branch of Human Auditory Nerve: Preliminary Report. Bull. Johns Hopkins Hosp., 54:208-210 (Mar.), 1934.

88. Dandy, W. E.: An Operation for the Cure of Tic Douloureux: Partial Section of the Sensory Root at the Pons. Arch. Surg., 18:687-734 (Feb.), 1929.

89. Dandy, W. E.: Certain Functions of Roots and Ganglia of Cranial Sensory Nerves. Arch. Neurol. & Psychiat., 27:22-29 (Jan.), 1932.

90. Dandy, W. E.: Ménière's Disease: Symptoms, Objective Findings and Treatment in 42 Cases. Arch. Otolaryng., 20:1-30 (July), 1934.

91. Dandy, W. E.: Effects on Hearing After Subtotal Section of the Cochlear Branch of the Auditory Nerve. Bull. Johns Hopkins Hosp., 55:240-243 (Sept.), 1934.

92. Dandy, W. E.: Treatment of Bilateral Ménière's Disease and Pseudo-Ménière's Disease. Tr. Am. Neurol. Assn., 61:128-133, 1935.

93. Coleman, C. C., and Lyerly, J. G.: Ménière's Disease: Diagnosis and Treatment. Arch. Neurol. & Psychiat., 29:522-537 (Mar.), 1933.

94. Swift, G. W.: Ménière's Disease. S. Clin. North America, 13:1465-1467 (Dec.), 1933.

95. Rutherford, R.: Auditory Nerve Section in Ménière's Disease. Brit. M. J., 1:660-661 (Mar. 27), 1937.

96. Walsh, M. N., and Adson, A. W.: (Unpublished data.)

VI

THE SIGNIFICANCE OF HOARSENESS

WALTER WELLS, M.D.

WASHINGTON, D. C.

When a symptom is one that has a definite and comparatively restricted indication, it may just as properly be made the subject of a medical discussion as the disease in which the symptom occurs.

Hoarseness is such a symptom. Unlike general manifestations such as fever, pain, etc., which arise from many causes, hoarseness occurs only when one particular organ, the larynx, is affected, and its presence is a sure indication of laryngeal trouble.

Furthermore, it is a symptom which must be accorded very special importance, because it is often the sole one present, and that, too, in cases in which the disease is one of extreme gravity.*

For the better understanding of the subject of this paper, let us briefly review the structure and function of the larynx, particularly as concerned in voice production.

For the larynx, we must bear in mind, is an organ of a double function. Primarily it was a part of the organ of respiration, and its sole function was to serve as a passageway for air to and from

---

*It must add, I think, to our thoughtful respect of hoarseness as a symptom when we recall that it was the one constant and outstanding symptom in the brief and tragic illness of our great first president.

Presumably in previous good health, Washington, after exposure to particularly inclement weather, rain, hail and snow falling alternately, was taken ill with a severe throat cold that ended fatally in the brief space of 48 hours. "The General," remarked his faithful biographer, Tobias Lear, "developed a hoarseness, which increased in the evening, but he made light of it." When papers were brought from the postoffice he sat up quite a while reading them, and when he met with anything particularly interesting he read it aloud, "as well as his hoarseness would allow." Between two and three o'clock that night he was awakened with a severe chill, and now it was observed that he could scarcely speak. To his hoarseness there was soon added difficult breathing and painful swallowing. The symptoms grew rapidly worse, despite all efforts to ameliorate them, and death followed from what his physician then called *cynanche trachealis*.

It was in all probability an intense streptococcic inflammation of the larynx, attended with edematous swelling, which closed the narrow opening behind the vocal cords, causing suffocation.

the lungs. But when speech was needed, nature, the great economist, instead of creating a completely new and separate organ, engrafted one upon the already existent respiratory tract which was admirably adapted for the purpose. At the top of the airway she set the vocal cords, and she utilized the lungs as a convenient bellows to throw them into vibration.

The larynx, considered as a musical instrument, may be classed as a double lipped reed of the oboe type. The vocal cords, or lips, are two narrow ligamentous bands, running horizontally from before backwards. In front they are fixed close together at the center of the receding angle of the thyroid; posteriorly they are movable, in a horizontal plane. When fully open for inspiration, they form two sides of a triangular space called the glottis; when brought together for the purpose of phonation they meet evenly in the midline. The in and out movement of the cords is made possible by an extraordinary arrangement of the arytenoid cartilages to which they are posteriorly attached. These are two small pyramidal-shaped cartilages, the bases of which form a capsulated articulation with the cricoid, and in such a way as to permit a combined in and out sliding, and at the same time a rotating movement about a vertical axis. To the anterior angles of the base of the little arytenoid the vocal cords are attached, and to the outer angles the minute muscles which run forward and backward to the cricoid cartilage just below.

When the backward running muscle (crico-arytenoideus postious) is contracted, the effect is to cause an outward swing of the front angle to which the cords are attached, that is, abduction or opening of the glottis; when on the other hand, the forward running muscle (crico-arytenoideus lateralis) contracts, the effect is to draw these points inward, thus bringing about the closing of the glottis by abduction of the cords.

While there is only the one muscle mentioned which serves the respiratory function by opening the cords, there are several which, by bringing the cords together and making them tense, serve the functions of voice production. All these except one are supplied by the same nerve—the recurrent branch of the vagus.

There is one, the action of which anyone can verify by placing the fingers against the larynx in the interval between the cricoid and thyroid cartilages, while intoning notes of varied pitch. This is the crico-thyroid and it is supplied by the superior laryngeal nerve. When a high note is sounded you can feel the cricoid cartilage drawn upward against the thyroid, putting the cords on a stretch and

increasing their tension, and by following with a low note an octave below, you can feel the cricoid dropping back into a low position, which relaxes the cords.

Very important for vocalization is the internal tensor (thyro-arytenoideus internus) muscle, the fibres of which run parallel with the cord and form a part of it. In fact, the vocal cords may be considered as the ligaments belonging to this muscle, and thus directly responsive to its contractions in sounding different notes. It is this highly developed internal tensor that gives the wonderful flexibility of the voice possessed by great singers.

The cords are two elastic bands, about three-quarters of an inch long in males and about half an inch in females, having normally smooth glistening white surfaces and even edges. As a result of disease they undergo a decided change in color, form and contour. The color may vary from pink or bright red in acute, to a dirty or slate-grey in chronic inflammation. The surface may become roughened and the edges uneven from exudate, infiltration, pseudo-membrane, ulceration or new growth, interfering with that nice approximation essential to the production of clear tones. Hoarseness can occur, however, even with normal appearing cords, as when the approximation becomes impossible because of a paralysis or by a growth which gets between them.

Above the true vocal cords and somewhat external, lies the so-called false cords. They are not normally intended for voice production, but do exceptionally come into play in certain kinds of unnatural shouting, or in case of paralysis of the cord or their destruction by ulceration. The effect is a disagreeable raucous voice, produced by obvious great effort.

A fairly brisk current of air from the lungs is needed to throw the vocal cords into vibration; there is, therefore, something in the general view that one must have good lung power to have a strong voice.

The cords can vibrate only when they are in more or less close apposition, these vibrations causing the stream of air to be chopped in a succession of pulsations which give rise to sound. When the cords are very tense and near together, the vibration frequency rises, producing high-pitched tones. When they are but loosely together, the tone is low pitched. The whisper is the result when the current of air passes between widely open cords, and is then whipped into speech by tongue, lips and teeth.

Though a whisper contains the essential elements of speech, it is lacking in the tonal qualities of the vibrating cord. It is, therefore, a sad substitute for vocal speech. For not only does it lack the carrying power often required, but it lacks emphasis and inflection, and the color of the voiced note. The vocal cords must come into play if the voice is to be expressive. The basic hum so produced, with its attendant harmonies emphasized in the resonant cavities of the nose, mouth and throat, give to it emotional qualities and musical charm.

It is sometimes necessary to warn our public speaking patients that mere loudness of the voice is not all that is needed to make themselves heard and understood. Clearness and intelligibility of speech depends to a great extent upon good articulation and accurate production and vowel resonance.

With proper vocal action, one can speak with little effort, yet be heard, for one thus gets full benefit of resonance. In this connection we might refer to the widespread belief that the deep or base register voice is due to chest resonance. The real purpose of the lungs is to serve as a bellows, and from a consideration of their physical qualities it is probable that they act as absorbers of sound rather than as resonators. As one writer expressed it, there is no more reason to expect the chest to have resonance than a bellows packed with a damp sponge.

To recapitulate, a good speaking or singing voice depends in the first place upon a good formation and healthy condition of the cords; second, upon a good development of the muscles that activate the cords; third, upon adequate lung power; fourth, upon a favorable development of the resonance cavities of the nose, mouth and throat; and fifth, upon proper methods of articulation.

The symptoms of hoarseness, though influenced more or less by all the factors of voice production, is essentially a trouble of the vocal cords. In former days when it was not possible to see the interior of the larynx during life, diseases of the vocal cords had to be diagnosed entirely by inference. Now, thanks to the arts of indirect and direct laryngoscopy, we can get a satisfactory view of the parts which are the seat of the disease.

Let us remind you at this point that practically all illustrations of the larynx by the indirect method are really of the image as seen reflected in the laryngoscopic mirror. As this differs from a view you would have by looking directly down upon the top of the larynx, you should know in what respect the difference exists. Re-

member that the image, and the illustration of it, is a view of the top of the larynx of the individual facing the observer. Therefore, the image as it appears in the mirror will show the epiglottis which is actually to the front as though it were to the back and farther away, and the arytenoids which are actually posterior, appear in front. The image in the mirror, you should know also, gives an illusory idea of the relative position of the true and false cords. They seem to be on the same level, whereas actually the true cords lie an inch below the false. The fore-shortening effect of the mirror furthermore gives a somewhat incorrect idea of the size of a growth situated on or about the cords, making it appear smaller or less extensive than it really is.

As hoarseness is a form of dysphonia due to an alteration involving the vocal cords, this alteration can for the most part be readily made out by laryngoscopic examination.

Leaving aside a few very rare diseases, we may briefly consider the following conditions in which it chiefly occurs: (1) Voice strain; (2) acute catarrhal laryngitis; (3) chronic catarrhal laryngitis; (4) acute edematous laryngitis; (5) acute spasmodic laryngitis (false croup); (6) diphtheria (true croup); (7) tuberculous diseases of the larynx; (8) syphilitic diseases of the larynx; (9) benign growths; (10) malignant growths.

Voice Strain: Hoarseness can develop as well from improper use as from excessive use of the voice. When it occurs suddenly after unusual exertion in the absence of other cause, we may justly say that it is due to strain.

Continuous improper use may not always be so evident, but should be suspected in those who complain of voice fatigue following singing exercises, or who speak in an unnatural cramped method, or who have poor resonance, and articulate indistinctly.

Objective changes are not always discoverable by the laryngoscopic examination. Violent strain may cause slight hemorrhages, usually shown as a red or later a brownish-yellow spot on the cord. In a chronically strained voice, we often note an elliptical space between the cords on abduction. This is due to paresis of the internal tensor muscle of the cord.

Singer's Node: A peculiar picture is sometimes seen in the larynx, consisting in the formation of small nodules on the edges of the vocal cords, which are directly traceable to improper use of the voice. They are small, rounded and uniform, partially trans-

lucent, projectious, often bilateral; found nearly always at the point of juncture of the anterior and middle third of the cord. They occur in singers who have been using improper methods, as for example, singing in a register beyond normal power, and producing tones in a way usually referred to as squeezing the voice. They seldom occur in those who sing regularly in the low registers, but mostly in tenors and sopranos.

They occur also in those who use the speaking voice badly and to excess, as teachers, who teach in dusty rooms, public speakers, who speak in the open, or individuals who are compelled to talk a great deal to deaf persons who do not use hearing aids.

Pachydermia Laryngitis: This is also a condition in which abuse of the voice is considered as a chief cause, for it is most often found in street-hawkers, show barkers and auctioneers. It is marked by the heaping-up of the tissue at the posterior extremity of the glottis, in the inter-arytenoid space and on the vocal processes.

It is due to excessive proliferation of pavement epithelium and multiplication of papillae. A thickening of the one cord is often accompanied by a crater-like depression on the corresponding location of the other cord. These patients usually have a husky, deep voice, a disagreeable sensation and a constant desire to clear the throat.

Laryngitis, Acute: There are persons in whom every cold has a tendency to attack the larynx. The voice becomes husky, with a frequent desire to clear the throat, and sometimes slight tenderness in the region of the larynx. Expectoration is generally scanty, but marked by the expulsion of small pearls or pellets of mucus. Examination of the larynx will show the laryngeal mucous membrane congested, and the cords normally white will appear pink, red or scarlet, and may be covered with shreds of mucus.

Laryngitis, Chronic: Repeated attacks of acute laryngitis eventuate in the chronic form. Even without the acute attacks, it is common in those who breathe habitually through the mouth, or who are exposed to dust, smoke, or irritating fumes, also in smokers who inhale, drinkers who use to excess strong alcoholic drinks, and in those subject to suppurative diseases of the sinuses. Mouth breathers will be discovered by a history of huskiness with tendency to increased clearing of the throat, particularly marked in the morning. In chronic laryngitis, there is a tendency to colds, accompanied by hoarseness. The cords will appear dull red, or dirty-grey, sometimes thickened and covered by sticky secretions.

Acute Edematous Laryngitis: This is an acute inflammation of the larynx, attended with edematous swelling of the membrane. When the swelling encroaches upon the glottis, difficult breathing is added to the hoarseness. In severe cases there is marked soreness of the larynx and painful swallowing. The condition is due to some virulent organism, generally the streptococcus. It is often preceded by a suppurative condition of the tonsil and peritonsillar tissue. Talking is painful, the voice is generally extremely hoarse, or may be aphonic. Examination shows the membrane swollen, partially translucent, sometimes red or purple. The swelling affects the epiglottis, arytenoid, or ary-epiglottic folds sometimes to an extent to render the anatomical landmark unrecognizable.

Acute Spasmodic Laryngitis (False Croup): This is a form of acute laryngitis peculiar to children. The little patient, hoarse during the day, wakes up suddenly at night with paroxysms of difficult breathing and cough, the cough being characterized by a brassy ring. The attacks are repeated successive nights with comparative freedom during the day.

Laryngoscopy is not usually possible, but if it were, an edematous condition would be seen in the region below as well as above the cords.

Diphtheria (True Croup): When diphtheria involves the larynx, we have what is generally denominated true croup.

The hoarseness and difficult breathing are due to the location of the pseudo-membrane in the larynx. The symptoms are present day as well as night, and unless checked by anitoxin, the dyspnea becomes gradually worse, accompanied by inspiratory stridor and chest retraction, cyanosis and asphyxia.

Although laryngoscopy is not generally used, it can be in a certain proporation of cases. I have had occasion thus to demonstrate the characteristic membrane in a number of cases in which none was visible in the upper throat, thus clinching the diagnosis before the culture report could be obtained.

Tuberculosis of the Larynx: The question of whether or not laryngeal tuberculosis is ever primary we would leave to those who take delight in academical discussion; the important practical fact is that we do sometimes have it, and in outspoken form, in patients in whom the pulmonary lesion is slight or indiscoverable.

We should think of its possible presence in any anemic, emaciated patient, who continues to cough or has frequent attacks of hoarseness. The dysphonia varies with location, character and extent

of the lesion, but the voice is generally described as being weak, muffled, veiled or toneless. In the advanced stage, there will be pain in swallowing, often referred to the ear. The pain is quite marked in the swallowing of liquids, which is the point of difference from the dysphagia of cancer, which is more marked in the swallowing of solid food.

The laryngeal picture in an early stage may show nothing more than an abnormally pale mucous membrane, with perhaps a little swelling in the inter-arytenoid region. Later you will see evidence of infiltration of the arytenoids with papillary vegetation in the intervening area. When the ulcerative stage is reached, the cords will be involved and appear red and irregularly serrated.

Syphilis of the Larynx: Syphilitic lesions, known to appear in such various forms and in so many parts of the body, do not escape the larynx. And when the cords are affected, as they are very apt to be, a hoarseness develops, regarded by some observers as rather characteristic. It is therefore called racedo syphilitica. The voice is harsh, raucous and presents a striking contrast with the voice of the tubercular, which, as we stated, is generally weak, muffled but painful.

It is characteristic in general of syphilitic diseases of the larynx that it is relatively painless as compared with other serious conditions as, for example, tuberculosis or cancer.

The form in which it most frequently occurs is the gummata. These are seen as smooth, round, reddish-yellow swellings. When they break down, ulceration follows, characterized by the deep, punched-out appearance, sharp edges, and surfaces covered by dirty looking pus or slough.

Benign Growth: Several kinds of benign growths occur in the larynx which may be responsible for a persistent hoarseness. The location of a growth has much to do with the occurrence of this symptom. A very small growth on the edge of the cords will produce considerable hoarseness, whereas a growth of greater size located elsewhere may be present for a long time without noticeable alteration in the voice.

Papilloma is the most common form of benign growth, especially in children in whom it develops to a size that will cause marked obstruction of breathing. It appears as a warty growth, pale pink in color, assuming generally a mulberry-like form.

The benign growths, named in order of their frequency, are: fibromata, myxomata, lipomata, chondromata. Cysts also occur, located generally in the epiglottis.

Cancer of the Larynx: The importance of hoarseness as a symptom cannot be better exemplified than in its occurrence in cancer of the larynx, for in this serious disease it is sometimes, in the early stages the only symptom present, and cancer is, of course, a disease in which early diagnosis is indeed of the utmost importance.

It is much more common in men than in women, and it is on this account that persistent hoarseness in elderly men may be very significant. The reason that hoarseness appears early is because laryngeal cancer of the intrinsic type has a predilection for the vocal cords, and because when a cord is thus involved, even though very slightly, infiltration of the underlying tissue affects its mobility. The reason that such a condition may persist quite a long time without other symptoms is due to the fact that cancer in this location shows little tendency to spread, owing to the isolated character of the intrinsic lymphatic circulation of the larynx.

On laryngoscopic examination we may see nothing more than a small greyish warty growth, or a small diffuse swollen area on the cord; but to the laryngologist, an appearance of this kind confined to one cord is very significant, more so if it is noted that the cord lags in movement as compared to its fellow of the opposite side.

A more typical form of epithelioma in the still early stage is a greyish fringe-like growth along the border of one of the cords. Later, when the neoplasm has broken down and the process extended, the diagnosis is unmistakable. The mirror now shows the presence of an irregular, sloughing growth; there is odor to the breath, intense pain in swallowing, emaciation and cachexia.

Laryngeal Paralysis: We might naturally expect the voice to be affected in cases of paralysis of muscles which serve the purpose of phonation. If, however, there is a complete paralysis of the abductors affecting both cords, instead of hoarseness there will be aphonia, because the cords cannot be approximated in a way to produce vibrations.

This is what we see in the hysterical form of aphonia, such as may occur following shock or emotional causes when the voice is suddenly reduced to a whisper, and later just as suddenly reappears.

When the external tensor, the one contraction of which can be felt on the front of the throat, is out of commission, we will see on the laryngoscopic mirror, as the patient attempts to phonate, a wave-like outline of the cords instead of a straight line. The voice is weak, feeble and easily tired.

The internal tensors, those directly connected with the cords, may be independently paralyzed as a result of local infiltration or severe acute inflammation or by voice strain. The voice in such a case is weak and toneless, and speaking is marked by so-called "phonetic waste," that is, escape of air between the insufficiently closed glottis. In the laryngoscopic mirror the picture is typical. In attempts at phonation the glottis takes the form of a narrow elliptical space.

Hoarseness may, also, be a symptom of a one-sided paralysis of the recurrent laryngeal nerve. This is not usually noted in the early stage but later on, when the paralyzed cord has assumed the cadaveric position and the sound cord attempts to compensate by passing over the midline to approximate it in the act of phonation.

This is easily recognized in the laryngeal mirror, when it is noticed that the paralyzed cord does not move with its fellow, and that on phonation you get an oblique instead of a straight midline between the cords.

In the foregoing discussion, we have assumed laryngoscopic examination as a matter of course. There are, however, cases in which laryngoscopic examination is not possible, as in children and adults with very irritable throats, and in other situations in which for one reason or another it is not immediately available. In spite of its absence, however, it may be worth our while to attempt to draw some inference based on the occurrence of the hoarseness which even in itself may sometimes afford valuable diagnostic data. For example, in an adult the development of a marked hoarseness in connection with an acute inflammation of the respiratory tract (an acute cold), permits us to infer with reasonable certainty the existence of an acute laryngitis.

In such a case, if in addition to a severe hoarseness there develops a difficulty in breathing, with soreness in the laryngeal region and painful swallowing, we are justified in assuming that the acute laryngitis is of an edematous nature, and there is reason for growing alarm because of the dangerous tendency to dyspnea. In a child, hoarseness in connection with an acute catarrhal condition of the upper air passages, accompanied by difficult breathing, may mean either diphtheria or spasmodic laryngitis. If the difficult breathing grows, day and night, gradually worse, it is probably diphtheria; if the symptoms subside during the day and return in paroxysmal form during the night it is probably spasmodic laryngitis, so-called false croup.

In a child who has hoarseness and difficult breathing unassociated with a cold and of long duration, we may rightly suspect the presence of laryngeal polyps, the most frequent benign growth in the young and one which sometimes increases rapidly in size to the point of obstructing breathing.

In young adults, say between the ages of 20 and 30, the presence of persistent and recurrent hoarseness, should make us think of a laryngeal tuberculosis. Especially would this be so if the individual is anemic and has a chronic cough.

Persistent hoarseness in persons beyond the age of 40 should always arouse suspicion of malignancy, at least if not due to evident mouth breathing or excessive alcoholism.

The character of the hoarseness differs and sometimes is quite characteristic. In laryngeal tuberculosis the voice may be described as weak, veiled or muffled, and painful with much use; in syphilitic laryngitis it is harsh and raucous, but painless. In bilateral tensor muscle paralysis it is hollow or toneless and easily tired; in unilateral cord paralysis it is unnaturally high pitched and easily tired.

From the standpoint of the laryngologist, diagnostic discussion of a laryngeal disease based solely on subjective symptoms is purely speculative, because the diagnosis cannot be considered as complete without a laryngoscopic examination.

However, speculative considerations are not without their value. They originate suggestions which tend to narrow the field of observation and to concentrate attention where attention particularly is needed.

The smart oil prospector looks for oil only when the right geological formation indicates its probable presence. Successful medicine depends in great measure on a knowledge of what to look for and where to look for it.

Doctors need to be reminded repeatedly of the indications of symptoms. Otherwise one is apt to hear too often such sad exclamations of disappointment and defeat as, "Oh, I never thought of that." A fuller knowledge of the meaning of hoarseness would no doubt more frequently lead to the discovery of its cause.

The prognosis will naturally depend upon the underlying cause, and in the case of the grave infections or malignancies resolves itself into a question of the general as well as local extent to the disease.

When the hoarseness is the symptom of a self-limited acute disease such as acute laryngitis, we may expect to see it clear up as soon as the disease runs its course.

In cases in which it can be rightly attributed to excessive or improper use of the voice, improvement will be in proportion to our success in restraining the excess or correcting the abuse.

When the hoarseness is due to a growth of any kind which prevents normal approximation of the cords, the prognosis for the voice is bad if the condition is not corrected, because of the extra strain put upon the laryngeal muscles in such case, in the effort to overcome the handicap. In the case, therefore, of singer's nodules due to voice strain, we have a real example of the so-called vicious circle. For the very thing that caused the trouble is in turn caused by it.

There is one kind of hoarseness that generally improves with time, that is, when it is due to vocal cord paralysis of one side; for after the paralyzed cord has finally settled down to a fixed position its immobility will be partially compensated by the overaction of the muscles of the other cord.

The hoarseness that we are all most frequently called on to treat is that due to an attack of acute laryngitis.

Outside of the general measures we adopt in treating any cold, there are some special indications for a cold in this location.

We believe in ice compresses, applied to the outside of the larynx, used within the first 48 hours, but not after that. During this early stage when the membranes are very sensitive we would not try to reach the interior of the larynx by astringent sprays or swabs. Later on mild sprays may be used, but none containing silver nitrate which is irritating to the larynx, and often produces severe glottic spasms. Soothing sprays may be used, and steam inhalation, containing benzoin, menthol and eucalyptol.

In acute laryngitis the prohibitions are more important than anything else. Smoking should be forbidden, especially inhaling. The patient should even avoid a room in which others are smoking, and any dust or fume laden atmosphere. It is important to avoid mouth breathing, and most important of all to avoid using the voice. This last is sometimes very difficult to enforce in the case of persons whose living depends upon their speaking or singing, and yet it is for these that it is particularly important.

A patient who is scheduled to give a lecture, or to go on in some theatrical act, will sometimes come to you and demand that you give him some spray or other treatment that will enable him to keep his engagement. Looking at the larynx, you see that the vocal cords are red and the whole laryngeal mucosa swollen and hyperemic. You know that to use the voice in such case is to increase and magnify the inflammation. You should, therefore, firmly forbid it, just as a surgeon would forbid the use of a tender, swollen joint in an acute arthritis. You should frankly tell the patient that there is no treatment, local or general, which under such circumstances will allow him to use his voice with impunity, and if he does, he runs the risk not only of prolonging the attack, but of becoming chronically hoarse. Chronic laryngitis and its attendant hoarseness is generally to be attributed to repeated and neglected acute attacks.

One should seek very carefully in such cases for a possible supporting cause in the condition of the nasal organ—as a suppurating sinus, with a constant draining of pus down the posterior wall of the pharynx in adults, and the presence of adenoids in children. Any nasal obstruction that induces the habit of mouth breathing should, of course, be corrected. Otherwise, the laryngeal mucosa will be kept constantly in an irritated state by the inhalation of dry and improperly prepared air.

Of course, you may meet sometimes, as I have, certain individuals who don't want to be cured of their hoarseness. I recall one very well known actress and one very well known actor, who prided themselves on their deep register, which they considered as inseparable from their stage personalities.

As a matter of fact, we have little doubt but that certain stage celebrities would in a measure lose their popularity if they lost their hoarse voices. Imagine the disappointment and disgust of the young gallery gods if Donald Duck were made to speak in clear musical tones. The gate receipts would, I fear, soon fall to the vanishing point.

Only a very few people have, however, a reason not to be cured of their hoarseness, for the hoarse voice is, generally speaking, a disagreeable voice; besides that it usually tires more easily than a normal voice, and grows worse with much usage. If it is one due to improper methods of voice production, the cure, of course, lies in the correction of those methods. This may require the individual to put himself or herself under the tutelage of a competent teacher. One can, in any case, do much for one's self by cultivating a natural

manner of speaking. One must leave the throat relaxed, instead of cramping the muscles, and practice careful distinct articulation on all occasions.

Finally, in certain cases, the cure or improvement in the hoarseness is of importance as an indication of recovery from the disease causing it. If hoarseness is due to a growth of any kind, it will never disappear until the growth has been in some way removed, either by direct or indirect laryngoscopy, or in the case of malignant diseases, it may be removed by an external operation.

We hardly need emphasize the vital importance of early removal of any malignant or partially malignant neoplasms. This is particularly true in the case of the larynx, because of the very distressful nature of the later stages of malignancy in this situation.

1606 Twentieth Street, N. W.

VII

CARCINOSARCOMA OF THE LARYNX*

IRA FRANK, M.D.

AND

MAURICE LEV, M.D.

CHICAGO

Since the time of Virchow various tumors have been described as carcinosarcoma, or malignant tumors of dual origin. Recently, Saphir and Vass,[1] and Saphir[2] have reviewed the literature of these tumors. From this study, and from the study of a series of their own cases, they have shed grave doubt as to the existence of such tumors. Rather, they strongly suggested that in most cases of diagnosed carcinosarcoma, the tumors were actually carcinomas which had undergone morphological variations and which at times resembled sarcomas.

In order to study further the validity of this interpretation, we undertook to study a large series of carcinomas of the same organ in order to see how frequently morphological variations approaching the sarcomatous arrangement of cells occurred within carcinomas and how many so-called carcinosarcomas we could find in any one organ. The organ chosen for initial study was the larynx because of the frequency of origin of atypical carcinomas from the respiratory tract.

The biopsies of 62 carcinomas of the larynx were studied microscopically by means of slides stained with hematoxylin and eosin. Where morphologic variations were noted, serial sections were cut through these tumors and stained with hematoxylin and eosin, and according to the method of van Gieson. Of these tumors 36 were diagnosed squamous cell carcinoma, 11 transitional cell carcinoma, 11 squamous cell carcinoma with transitional cell features, and 4 transitional cell carcinoma with squamous cell features. Throughout these tumors an attempt was made to notice those areas where the epithelial arrangement of tumor cells was disturbed, with a tendency towards the sarcomatous arrangement—that is a whorled con-

*From the Department of Pathology of the Nelson Morris Institute of the Michael Reese Hospital, Chicago, Illinois. Aided by the Nelson Morris fund.

figuration of spindle shaped cells which when viewed by itself without regard to the obvious epithelial features could be interpreted as a malignant connective tissue tumor.

Our results are tabulated in the accompanying table one. It will be noted that 48.4% of all carcinomas of the larynx in this series showed some evidence of this atypical arrangement. The most common type of tumor that showed this was the transitional cell carcinoma. However, the fact that 44.4% of squamous cell carcinomas in this series likewise showed a similar tendency shows the great potentiality in all types of carcinomas of the larynx for an atypical "sarcomatous" arrangement of cells.

TABLE I

THE "SARCOMATOUS" TENDENCY IN CARCINOMA OF THE LARYNX

Type of Carcinoma	Total No.	No. Showing "Sarcomatous" Features	Pct. Showing "Sarcomatous" Features
Squamous cell	36	16	44.4
Transitional cell	11	7	63.6
Predominantly squamous cell with transitional cell features	11	7	63.6
Predominantly transitional cell with squamous cell features	4	0	0
Total	62	30	48.4

In still further studying these atypical tumors, it was noted that in any one tumor the tendency towards sarcomatous arrangement occurred in two types of cells,—the transitional and the squamous cell. In the case of the transitional cell, this tendency became manifest by the cell becoming markedly elongated, spindle shaped and arranged in whorled formation as in a "stroma". This occurred (A) where the tumor was composed completely of transitional cells, (B) where separate groups of transitional cells were found accompanying a predominantly squamous cell carcinoma, and (C) where in a predominately squamous cell carcinoma the periphery of the squamous cells going into the formation of pearls consisted of transitional cells. In the latter case, it would seem that these transitional

cells were compelled to become elongated and spindle shaped by compression between adjacent and apparently less rapidly invasive groups of squamous cells. Here there was no question of the identity of the cells, as they were seen to be continuous with the normal looking transitional cells, and their accumulation in any one field was always accompanied by distinct epithelial elements. However, in (A) and (B) large fields of elongated transitional cells very often resembled sarcoma when viewed by themselves, but adjacent fields revealed their true identity. This degree of tendency toward the sarcomatous arrangement varied from biopsy to biopsy (Figs. 1 and 2).

In the most advanced case (Fig. 2), almost the entire transitional cell carcinoma seemed to possess a sarcomatous arrangement and only multiple sections revealed the identity of the fundamental cell as being epithelial. This type of tumor, on superficial examination, could very well be classified as carcinosarcoma. However, when viewed from the standpoint of our series of similar tumors, we cannot help but realize that this tumor represents to an extreme degree what is seen in over half the transitional cell carcinomas, and that we are dealing with a peculiar type of transitional cell carcinoma.

In the case of the squamous cell, the tendency towards the sarcomatous arrangement was found in one of the two types of squamous cell carcinomas into which all the squamous cell carcinomas could be classified. In one type, blocks of squamous cells going into pearl formation were pocketed within the stroma. The border between the stroma and the peripherally lying squamous cells was sharp. Here no tendency towards sarcomatous arrangement could be found. In the second type (Figs. 3 and 4), however, there was no sharp line between the squamous cells and the stroma. Instead, there was a halo about each pearl where the peripherally lying squamous cells, taking on an elongated spindle form, invaded the stroma. These cells still retained their squamous cell staining characteristics. The result is that a new "stroma" was found containing both epithelial and connective tissue cells. Where this was present in a mild degree (Figs. 3 and 4) the identity of the tumor was not in question, although a van Gieson stain was at times necessary to establish the nature of the halo about the epithelial pearls. However, various tumors showed this tendency in various degrees (Figs. 3-6), and where it became very marked (Fig. 6) the appearance of the stroma formed by the epithelial and connective tissue spindles could very well be mistaken for "sarcoma". As a matter of fact in a few instances most of the tumor consisted of just this kind of "combined

stroma", which, with the occasional epithelial pearls, could very well be interpreted as carcinosarcoma. The study of these tumors, with the other tumors manifesting similar characteristics but to a less degree, reveals them to be exclusively of epithelial origin—simply squamous cell carcinoma.

REVIEW OF THE LITERATURE OF CARCINOSARCOMA OF THE LARYNX

The above analysis of a fairly large series of carcinomas thus clearly shows that certain carcinomas can resemble sarcomas. This, therefore, raises the question: are the carcinosarcomas reported in the literature true tumors of dual origin or are they simply carcinomas which take on the appearance of sarcoma as in our above cases.

The pertinent clinical and pathologic details of five cases of carcinosarcoma of the larynx found in the literature are presented in the accompanying table II. The photomicrographs of these cases (where presented by the author) are found in the accompanying illustrations (Figs. 7-10).

In the case of Szmurlo[3], there were two different types of growth seen grossly. Multiple papillary excrescences were present on the vocal cords and the subglottic region. Histologically, these proved to be carcinoma, according to the author. In addition, there was a bean-shaped sessile tumor on the vocal cord which histologically proved to be sarcoma, according to the author. The actual histological features of this tumor are not available to us, and therefore, we cannot judge for ourselves the nature of this tumor. However, from the author's own admission, there was no mixture of the two types of tumor histologically and he believed that he had both a carcinoma and a sarcoma growing in different parts of the larynx. Also we question very much the diagnosis of sarcoma from the description of the histologic features of the bean-shaped tumor. It could very well be a fibroma or granulation tissue with giant cells, especially in view of a 40-year history of voice difficulty.

The case of Kahler[4] is a tumor of the pyriform sinus which had produced metastases to the cervical lymph nodes, pleurae and spinal column. The primary tumor histologically was considered carcinosarcoma, while all the metastases showed squamous cell carcinoma. In Fig. 7, we note what was considered by the author to be sarcoma. We may compare this to our own Fig. 2. Obviously in both of these cases we are dealing with transitional cell carcinomas which show a moderate to marked degree of "stroma-like" formation.

In the case of Ullmann[5], a pedunculated tumor of the vocal cord was considered carcinosarcoma. Fig. 8 (A, B, C) are reproductions of some of the author's photomicrographs of this tumor. If we compare these with Figs. 5 and 6, representing two of our squamous cell carcinomas with marked "sarcomatous" tendency, we see that the three tumors are identical in type. We are dealing in all three cases with malignant squamous cells with a marked tendency towards the formation of a combined stroma. In our above analysis, we have shown this to exist in about 50% of squamous cell carcinomas, which tendency can be traced from its mildest to severest form in Figs. 3-6. The tumor of Ullmann and our case 2 represents a most marked degree of this tendency.

In the case of Ricci[6], a pedunculated polyp was removed from a vocal cord, and was considered histologically to be a sarcoma. Nine years later, a recurrent polyp was removed from the same vocal cord which was considered histologically to be carcinosarcoma. In Fig. 9 we see reproductions of photomicrographs of Ricci's case. The squamous cell carcinomatous elements are evident. But what the author has considered sarcoma (Fig. 9C) is apparently benign connective tissue. The malignant cells arranged in "stroma-like" formation are carcinoma cells, which have undergone morphological variations. In other words, we are dealing with a squamous and transitional cell carcinoma invading a previously existing benign connective tissue formation, whether it be fibromatous or granulomatous, with the formation of a combined stroma consisting of elongated squamous and transitional cells and fibroblasts.

In the case of Schwartz[7], a polypoid tumor considered grossly a lipoma was found histologically to be a carcinosarcoma. Fig. 10 is a reproduction of Fig. 2 of his photomicrographs. He does not clearly state whether this figure represents his case of carcinosarcoma of the larynx but it is so implied. We may compare this with Fig. 8 of Ullmann's case, and Figs. 5 and 6 of our own series. In the case of Schwartz we are apparently dealing with a squamous cell carcinoma with a marked tendency towards the formation of a combined stroma consisting of elongated squamous cells and benign connective tissue elements.

Thus a review of the literature reveals five cases of carcinosarcoma of the larynx to have been reported. An analysis of three of these cases and a comparison of these cases with some in our series of carcinomas show a marked similarity between these so-called carcinosarcomas and our proven carcinomas. In the cases of Szmurlo and of Ricci, with which we have no comparison, an analysis of the

TABLE II

SUMMARY OF CASES OF CARCINOSARCOMA OF THE LARYNX REPORTED IN THE LITERATURE

Author	Sex	Age	History	Gross Description of Laryngeal Pathology	Microscopic Description	Our Interpretation
Szmurlo 1894	M.	50	Voice difficulty since 10 years of age.	Both false vocal cords red, swollen, with papillary excrescences. Both true vocal cords red and thickened with ulcerations. Movement of cords normal. Subglottic region also reddened with papillary excrescences. Bean shaped sessile tumor-like mass near free end of right vocal cord projecting beyond free end.	Bean shaped tumor showed giant and spindle cells. Considered sarcoma. Excrescences showed cords of epithelial cells with cornification extending down in normal stroma, considered carcinoma. Considered two separate tumors—one carcinoma, one sarcoma.	No histologic pictures available. Most likely a carcinoma and a fibroma in separate areas.
Kahler 1908	M.	68	Difficulty in swallowing for three months. Developed pain in arms with paralysis. Patient died of bronchopneumonia six months after onset.	Autopsy showed walnut sized, flat polypoid tumor behind left aryepiglottic fold in left pyriform sinus; metastases to cervical lymph nodes, pleurae, spinal column.	Biopsy of tumor showed pleomorphic giant cell sarcoma. Study of tumor post mortem showed masses of epithelial cells considered basal in some areas, and in others elongated spindle cells considered sarcoma. All metastases showed squamous cell carcinoma.	Photomicrographs reveal transitional cell carcinoma with morphologic variations.
Ullmann 1922	M.	60	Hoarseness, four months. Difficulty in breathing, one week. No cervical glands palpable.	Cherry sized greyish red smooth firm pedunculated tumor 2.5x1x1 cm. on free end of right vocal cord near processus vocalis. No ulceration or erosion. Tumor removed with snare. One week later biopsy of thickened left vocal cord showed no tumor. Received radium. Six weeks later small ulcerated prominence near right processus vocalis seen. Six weeks later right vocal cord irregular. Further radium treatment given. Died from unrelated intercurrent infection.	Nests of atypical squamous cells with epithelial pearls. Considered squamous cell carcinoma. Stroma in which these nests lay consisted of intertwining and whorled spindle cells with pleomorphic nuclei with many mitotic figures; occasionally giant cells, many plasma cells, few round cells, little vascularity. Some regions showed epithelial pearls covered by coat or sarcoma cells.	Photomicrographs show squamous cell carcinoma with marked tendency towards the formation of a "combined stroma" consisting of elongated squamous cells and fibroblasts.

TABLE II—Continued

Author	Sex	Age	History	Gross Description of Laryngeal Pathology	Microscopic Description	Our Interpretation
Ricci 1933	M.	54	Patient first seen 9 years previously with hoarseness. Tumor removed easily and patient was well for 9 years. Returned again and similar tumor found which was again removed with difficulty. Five months later more diffuse tumor removed with difficulty.	When first seen, pedunculated polyp on left vocal cord. Nine years later similar polyp on left vocal cord. Five months later diffuse involvement of whole vocal cord.	First polyp considered sarcoma with giant cells. Second tumor showed pockets of malignant squamous cells lying in stroma consisting of malignant spindle cells with giant cells.	Photomicrographs show squamous and transitional cell carcinoma infiltrating into (preexisting) fibroma or granulation tissue with formation of a combined stroma of elongated squamous and transitional cells and fibroblasts.
Schwartz 1936	M.	56	Hoarseness for one year. Tumor removed. Died one year later from heart failure. No autopsy.	Apparently polypoid tumor clinically considered lipioma.	Nests of squamous cells with cornification lying in the midst of malignant spindle cells considered sarcoma. Author states that two types of cells are sharply demarcated.	If Fig. 2 typifies the tumor, then it is a squamous cell carcinoma with marked tendency towards combined stroma of squamous and fibroblastic cells.

description in the former and a study of the photomicrographs in the latter make very unlikely the presence of sarcoma in these tumors, and we believe them to be carcinomas invading either fibrous or granulation tissue.

FACTORS RESPONSIBLE FOR MORPHOLOGIC VARIETIES IN CARCINOMAS

We are thus confronted with the fact that a certain number of carcinomas of the larynx are morphologically atypical, in areas closely resembling sarcomas. The question thus arises what are the factors which are involved in the production of these tumors? Is the tendency towards the formation of a combined stroma in squamous cell carcinomas, or the tendency towards spindle-shaped elongation with whorled formation in cells of the transitional cell carcinoma a property of the tumor itself, a property of the environment of the tumor, or a function of both? And what is the relationship of radium to this tendency in carcinomas of the larynx?

As far as irradiation is concerned, a comparative analysis of the records of 19 cases showing "sarcomatous" tendencies with 19 cases which did not show this tendency revealed the following: (1) Only one of the "sarcomatous" group had received radium: (2) Five of the "non-sarcomatous" group had received radium and yet did not show any "sarcomatous" tendencies. The one case, however, among the former group, which had been irradiated, showed the most marked tendency towards "sarcomatous" arrangement. The interpretation of these facts can only be that radium per se is not primarily responsible for the "sarcomatous" tendency of carcinomas of the larynx. Its possible secondary role by aiding in the production of fibrosis will be later discussed.

What is the role of chronic inflammation in the production of this tendency? An analysis of our two groups of cases shows that there is just as much and in some cases more chronic inflammation in the typical carcinoma as in the atypical. And in addition, we have a number of cases with "sarcomatous" tendencies which do not show any chronic inflammation at all. Therefore, chronic inflammation, per se, is not primarily involved in the "sarcomatous" arrangement of carcinoma cells. Its indirect role in the production of fibrosis will be later discussed.

The influence of connective tissue, however, in the production of the "sarcomatous" tendency, becomes very obvious in our study of these two groups. In the transitional cell carcinoma this is apparently a less important factor than the tendency towards a whorled

arrangement of the spindle shaped carcinoma cells themselves. However, in the squamous cell carcinomas, there is definitely a much greater amount of connective tissue in the atypical carcinomas, and this tissue is denser and more fibrous. In the typical carcinomas, the connective tissue is generally more delicate and less in amount. Thus, in more superficial biopsies, there is generally less atypical arrangement than in sections penetrating the deeper layer of the larynx in the region of denser fibrous tissue, smooth and striated muscle and cartilage. Also the greatest "sarcomatous" tendencies were found in carcinomas penetrating scar tissue. We have two such cases. A biopsy of the primary tumor, in each case, showed a mild tendency towards atypical arrangement. About a year later, after laryngectomy, a biopsy through the scar tissue around the tracheotomy wound showed a typical "carcinosarcoma". This is again seen from the study of the literature, in Ricci's and in Szmurlo's cases. In the former there was a carcinoma invading granulation tissue which at removal nine years previously was called sarcoma. And in the latter, in a patient who had been troubled with his voice for 40 years, a carcinoma had probably invaded either a fibroma or scar tissue. Thus it is apparent, that the connective tissue environment, either previously present, or reactive to the carcinomas, is greatly responsible for the "sarcomatous" tendencies of a carcinoma. If the connective tissue is the reaction to the carcinoma, whether the amount of connective tissue is a function of the defense mechanism of the host or a property of the carcinoma itself, our present study obviously cannot prove. It is apparent that in so far as irradiation or chronic inflammatory changes lead to fibrosis, so will the further growth of a carcinoma be influenced.

However, that the connective tissue environment is not the sole cause of the "sarcomatous" tendencies in squamous cell carcinoma, is seen by the following facts. A few of our carcinomas show a moderate to marked tendency towards atypical arrangement lying in a loose stroma or a very scant stroma, where there is very little "combined stroma" and where the "sarcomatous" tendency is in the carcinoma cells themselves. That this is true is substantiated by the fact that certain of our carcinomas lying in dense stromas still show the "sarcomatous arrangement" only to a mild degree. Thus, it is evident that the tendency towards stroma-like arrangement in many squamous cell carcinomas is also a property of the tumor itself just as in the transitional cell carcinoma.

In summary, the sarcomatous tendency of transitional cell carcinomas lies in the tendency towards a whorled arrangement of

spindle shaped cells, which may become aggravated by growth into connective tissue. Many squamous cell carcinomas likewise have this tendency which is inherent within the tumor cells themselves. This tendency in the squamous cell carcinomas, however, becomes greatly increased as the connective tissue environment increases. Thus the tumor appears to be a "carcinosarcoma".

CLINICAL SIGNIFICANCE OF THE "SARCOMATOUS" ARRANGEMENT

The final question and the most important one is: Does this tendency towards "sarcomatous" arrangement in carcinoma indicate a greater tendency towards malignancy? A priori, two points of view may be entertained. The first is that this characteristic implies a greater invasive tendency. The other is that the greater amount of connective tissue seems to indicate a greater resistance on the part of the patient. An answer to this question necessitates a comparative study of the longevity of life in patients with tumors with or without this characteristic, the tendency to metastasis, the tendency to recur after various procedures for removal, the location of the tumor, the type of cell, the degree of anaplasia, and the number of typical and atypical mitotic figures. Unfortunately this study cannot answer the question fully. Many of the biopsies were from outpatients in which cases the history and subsequent course is at present not obtainable. Also the exact location of the tumor is sometimes not recorded.

However, certain suggestive information is available. Tumors showing a more marked sarcomatous tendency had in some cases a prolonged history of hoarseness, in one case going back twenty-five years. This again recalls the cases of Szmurlo and of Ricci. Again from a laryngoscopic standpoint, these tumors had a greater tendency to secondarily involve the arytenoids than did the other. As far as metastasis is concerned, this study shows that the most important factor is primarily the location and not the type of tumor. Thus of 25 cases of squamous cell carcinoma, both typical and atypical, four showed metastases to the lymph nodes in the neck. Of seven transitional cell carcinomas, one showed metastases to the regional nodes. However, of the five tumors that metastasized, three involved the arytenoids, one involved the epiglottis, and the exact location of the last is not clearly stated. Thus, taking clinical data as evidence, it would seem that a tumor involving the vocal cords alone hardly ever metastasizes, and it is the location of the tumor which is the most important factor in metastasis. The tendency towards "sarcomatous" arrangement is apparently not concerned

in the question of metastasis. One would expect its role in recurrence to be great, but our study does not offer the necessary data to reach a definite conclusion.

SUMMARY

1. Histologic sections from 62 squamous and transitional cell carcinomas of the larynx were studied with the objective of analyzing the "sarcomatous" features in these tumors.

2. It was found that almost half of the squamous and more than half of the transitional cell carcinomas showed this tendency to a variable degree. Where it was most marked, the tumors resembled what has been called carcinosarcoma.

3. Five cases of carcinosarcoma of the larynx found in the literature were reviewed, analyzed and compared to our proven carcinomas. They were found to be almost identical with our tumors, and therefore, they are not carcinosarcomas but carcinomas which have undergone morphological variations.

4. The factors responsible for the sarcoma-like arrangement of cells are two:

(1) An inherent tendency of the tumor itself.

(2) The amount of connective tissue environment of the tumor.

5. The question of the clinical significance of the "sarcomatous" features in these carcinomas is not definitely answered in the present study.

104 S. MICHIGAN.

BIBLIOGRAPHY

1. Saphir, O., and Vass, A.: Carcinosarcoma. Am. J. Cancer, 33:331, 1938.

2. Saphir, O.: Morphologic Variations of Tumor Cells. Am. J. Path., 14:443, 1938.

3. Szmurlo: Ein Fall von Coexistenz von Sarkom und Carcinom im Kehlkopf. Medycyna, 29, Jahrgang, Warschau, 1894. Quoted by Ullmann.

4. Kahler, O.: Ein Carcino-Sarcom des Recessus piriformis bei Ekchondrose des Ringknorpels. Deutsche med. Wchnschr., 34:644, 1908.

5. Ullmann, H.: Ein echtes Carcinosarkom des Kehlkopfes. Ztschr. f. Hals-, Nasen-u. Ohrenh., 1:130, 1922.

6. Ricci, B.: Carcinosarcoma di Una Corda Vocale. Oto-rino-laring. Ital., 3:259, 1933.

7. Schwartz, H.: Über Carcinosarkome. Frankfurt. Ztschr. f. Path., 49:247, 1936.

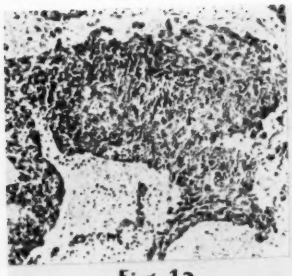


Fig. 1a

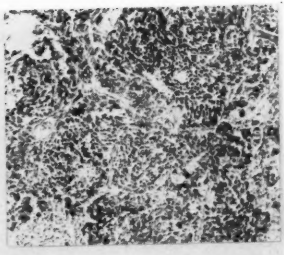


Fig. 1b

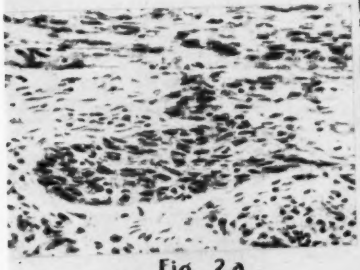


Fig. 2a

Fig. 2b

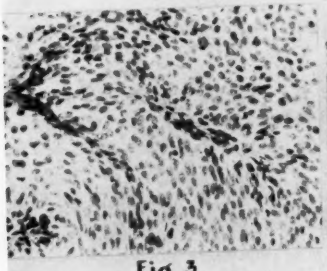


Fig. 3

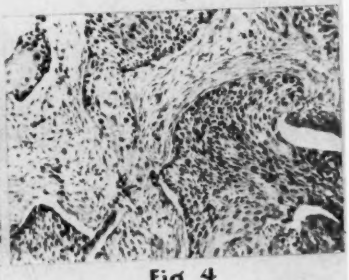


Fig. 4

Fig. 1. Transitional cell carcinoma of the larynx with a moderate tendency toward "sarcomatous" arrangement. Hematoxylin-Eosin Preparation x115.

A. Obvious transitional cell features.

B. Portions of the tumor showing a moderate tendency toward "sarcomatous" arrangement.

Fig. 2. Transitional cell carcinoma of the larynx with a marked tendency toward "sarcomatous" arrangement. Hematoxylin-Eosin Preparation x280.

A. Obvious transitional cell features. Note the tendency toward the spindle-shaped epithelial cells intermingling with the stroma.

B. Marked "sarcomatous" features. It is difficult morphologically to state whether the malignant cells are of epithelial or connective tissue origin.

Fig. 3. Squamous cell carcinoma of the larynx with a slight tendency toward "sarcomatous" arrangement. Hematoxylin-Eosin Preparation x220. Note the tendency of squamous cells to become spindle shaped as they are crowded between adjacent groups of cells.

Fig. 4. Squamous cell carcinoma of the larynx with a moderate tendency toward "sarcomatous" arrangement. Hematoxylin-Eosin Preparation x105. Note the tendency toward the formation of a combined stroma consisting of spindle shaped squamous cells and connective tissue cells.

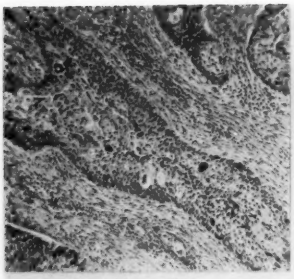


Fig. 5a

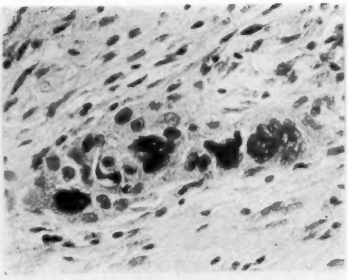


Fig. 5b

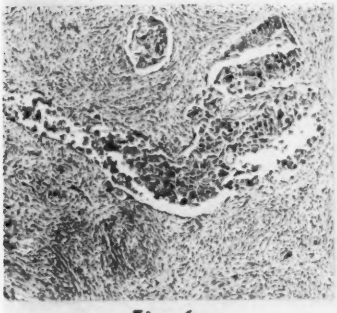


Fig. 6a

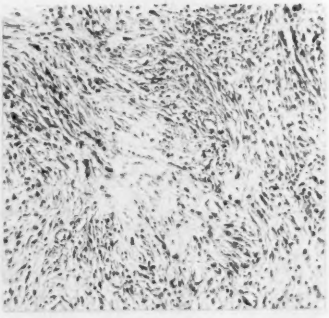


Fig. 6b

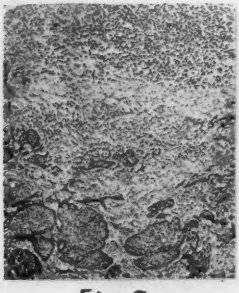


Fig. 7a

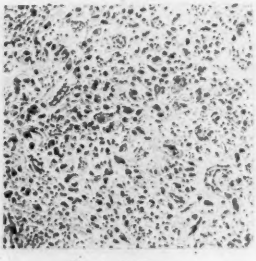


Fig. 7b

Fig. 5. Squamous cell carcinoma of the larynx with a marked tendency toward "sarcomatous" arrangement. Iron Hematoxylin-Eosin Preparation.

A. Note the tendency toward the formation of a "combined" stroma. The obviously epithelial elements, however, still predominate.

B. Note the lack of a clear line of demarcation between the obviously epithelial elements and the "combined" stroma.

Fig. 6. Squamous cell carcinoma of the larynx with marked tendency toward "sarcomatous" arrangement. Hematoxylin-Eosin Preparation.

A. Note the large amount of "combined" stroma which exceeds the epithelial elements in this tumor. Tumors of this kind have been called "carcinosarcoma" in the literature.

B. Showing the stroma which could very well be mistaken for sarcoma.

Fig. 7. Reproductions of the illustrations of the case of Kahler.

A. Showing both the carcinomatous and sarcomatous elements of the tumor, according to the author. According to our interpretation the so-called sarcoma cells are transitional cells which have undergone morphologic variations.

B. Showing what Kahler considered sarcoma. In our interpretation this is an atypical transitional cell carcinoma. (Compare with Fig. 2.)

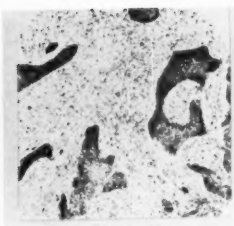


Fig. 8a

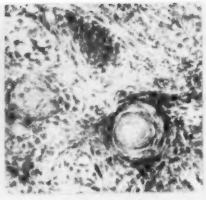


Fig. 8b

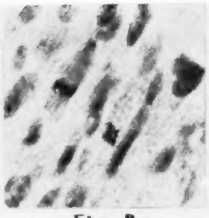


Fig. 8c

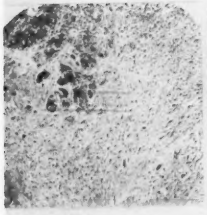


Fig. 9a

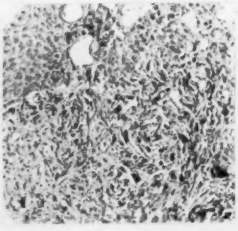


Fig. 9b

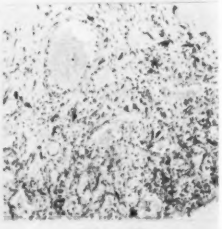


Fig. 9c

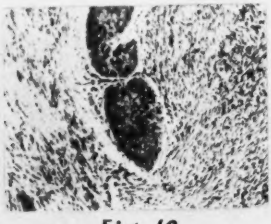


Fig. 10

Fig. 8. Reproductions of the illustrations of the case of Ullmann.

A. Low power view of tumor showing both the carcinomatous and the sarcomatous elements of the tumor, according to Ullmann. In our interpretation, the stroma seen here is a "combined" stroma consisting of both epithelial and connective tissue cells. (Compare with Figs 5 and 6.)

B. Higher magnification. The stroma seen here is considered sarcoma by the author. In our interpretation, this is a typical example of the tendency of squamous cells to "wander" into the connective tissue, producing a "combined" stroma. (Compare with Fig. 5B.)

C. High magnification showing sarcoma cells, according to Ullmann. In our interpretation, this shows the marked tendency of epithelial cells to resemble sarcoma cells in some tumors.

Fig. 9. Reproductions of the illustrations from the case of Ricci.

A. Section of tumor showing epithelial elements. Note that the tumor is growing into much scar tissue. Similar carcinomas in our series showed similar characteristics.

B. According to the author, this section shows carcinoma and sarcoma in close proximity. In our interpretation the squamous cells have invaded connective tissue and resemble sarcoma cells.

C. This section, according to Ricci, presents the sarcomatous features. In our interpretation, this is granulation tissue with a possible scattering of carcinoma cells.

Fig. 10. Reproduction of the illustration of the case of Schwartz. The author considers the "stroma formation" here as sarcoma. In our interpretation, this is simply a "combined" stroma of carcinoma and connective tissue cells.

VIII

EXPERIMENTAL HYPERPARATHYROIDISM AND OTOSCLEROSIS*

EARL C. SLAUGHTER, M.D.

BOSTON

In reviewing the etiology of otosclerosis as given by various writers, the number of possible causes is astounding. However, in this maze of causative factors the phrase "abnormalities of the ductless glands" appears with unusual consistency. Included in this category is hyperparathyroidism. During the past few years several observations have been made as to the cause of otosclerosis, but in a search through the literature nothing is to be found as to experimental work carried out in an effort to produce the pathological picture of otosclerosis.

SUMMARY OF OPINIONS AS TO CAUSE

Brule believes clinical otosclerosis and osseous ankylosis of the stapes are identical. He attributes otosclerosis to irritative factors involved in the movements of the foot plate of the stapes and the pull of the tensor tympani in the opposite direction, especially in individuals predisposed by heredity to hyperostoses in this region.

Wittmaack believes venous stasis is the causative factor in the production of otosclerosis.

Morris Weber, Jr. regards otosclerosis as a localized osteitis fibrosa with a local metabolic dysfunction as an etiologic factor. There is a general agreement that a probable hereditary factor is present in most cases. It also is said to occur more frequently in the female than the male. Other causative factors which have been mentioned are anemia, syphilis, fragility of the bones and pregnancy.

In a recent article Cody[1] has said, "The clinician by noting the endocrine type of the patient or the effect of glandular therapy has found grounds for the faith—for faith it still largely remains—that otosclerosis in many patients is basically a form of endocrinism.

---

*From the Mosher Laboratory of the Massachusetts Eye and Ear Infirmary. This subject was investigated at the suggestion of Dr. H. P. Mosher.

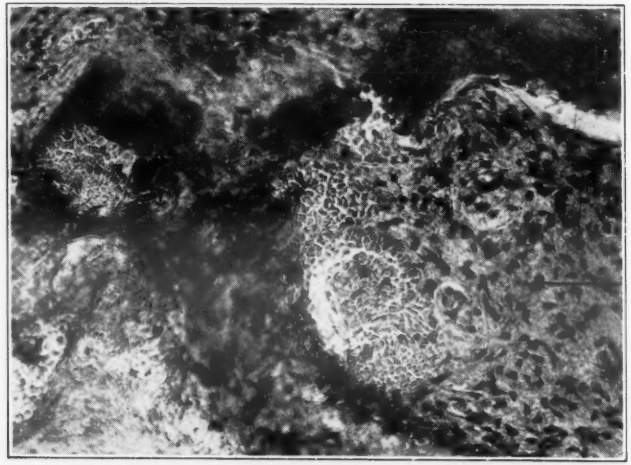


Fig. 1. Osteitis Fibrosa Cystica. Histologically, cases in this group show spicules of newly formed fibrous bone with active osteoclasts and simultaneous bone resorption by giant cell osteoclasts. The mobilization and excretion of calcium deplete the bone spicules and apparently resorption by giant cells complete the process.

The facts which support this view are that otosclerosis is frequently a familial disease, that the majority of occurrences are in women and that in women the onset or the exacerbation of the symptoms often coincides with puberty, menstruation, pregnancy and the menopause—the results of endocrine functions."

With the foregoing statements in mind it seemed logical to assume that experimental work in the field of metabolic dysfunctions would lead to more knowledge as to the cause of the pathological picture seen in otosclerosis. Since osteitis fibrosa (von Recklinghausen's disease), osteitis deformans (Paget's disease),[2] and otosclerosis are much alike in their pathological changes, and since osteitis fibrosa and osteitis deformans are caused by abnormal calcium metabolism, it was thought that through the production of experimental hyperparathyroidism the bony labyrinthine capsule could be studied for possible changes, and their relation to otosclerosis.

Osteitis fibrosa has been produced experimentally by the injection of parathyroid hormone.[3] In this experiment it was decided to use the newer form of parathyroid hormone, namely, A.T. 10* (Anti-Tetany).

Recently 19 cases of otosclerosis were reported in which parathyroidectomy was performed as a therapeutic procedure. In all cases there was a marked improvement in the hearing followed by a decrease, but the end-result was improvement in hearing, more especially for the higher tones. It is worthy of note that in this series eleven cases were women.[4]

To carry out the observation of the effect of parathyroid hormone (A.T. 10) on the labyrinthine capsule of the ear, the drug was given to guinea pigs, rabbits and dogs. In general, young animals are more susceptible to the effects of the hormone. Also the serum calcium of herbivorous animals, such as rats, mice, rabbits and guinea pigs, responds much less readily to the hormone, and the postmortem picture so characteristic of its effects on dogs and cats is not seen. In herbivorous animals, on the other hand, repeated doses cause the deposit of calcium in the soft tissues, particularly the arter-

*This is also known as dihydrotachysterol. Like Vitamin D, it is a photochemical derivative of ergosterol. It was developed by Holz, 1936, in Berlin, for the treatment of hypoparathyroidism. That one can easily raise the calcium of the blood to any desired level with this substance requires no further testimony. The literature was reviewed by Albright, Bloomberg, Drake and Sulkowitch, in a paper which contrasted the action of dihydrotachysterol with that of Vitamin D.

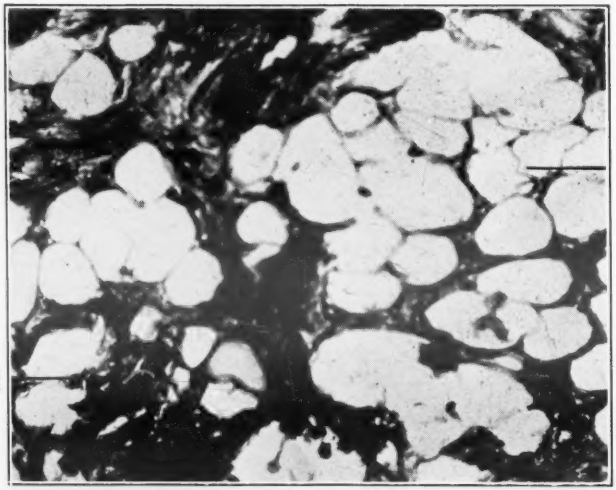


Fig. 2. Paget's Disease of the Skull. Histologically the basis of the disease is bone absorption replaced by ossification. Many giant cell osteoclasts may be seen destroying old spicules of bone, while at the same time there is an increase of loose connective tissue.

ial tree.[5] This is infrequent in dogs. In the human, hypercalcemia is produced nearly as easily as it is in dogs. In both men and dogs tolerance to the hormone not infrequently becomes established after a certain number of doses. For this reason dogs were used in greater number. Also the hormone was given over a period of time and then its administration stopped for a short period, in the hope that a tolerance would not be established and the condition of hypercalcemia constantly maintained.

Chute points out that, during the past eight years at the Massachusetts General Hospital, 36 cases of hyperparathyroidism were admitted. Women were affected more than twice as often as men. The predominating complaints in more than half of the cases were of urinary origin in the form of calculi. After removal of the parathyroids the formation of the calculi ceased.

During hypercalcemia the bony trabeculae showed large numbers of osteoclasts, which were believed to be responsible for the removal of the calcium. When the animal became tolerant to the hormone the osteoclasts were replaced by osteoblasts and the density increased; that is, calcium was deposited. It has been shown by several workers that either parathormone or irradiated ergosterol give rise to metastatic calcification (urinary tract, muscles and tendons, ligaments) and to bony changes analogous to osteitis fibrosa.[6] In pregnancy, negligible amounts of calcium are deposited in the skeleton before the fifth month of intra-uterine life, and over 60 per cent of the skeletal calcium of the new-born is the result of deposition during the last two months of prenatal life.[7] In other words, there is a calcium retention or a positive calcium balance, which can be likened to the hypercalcemia induced by the administration of the hormone.[8]

Hyperparathyroidism and osteitis fibrosa are synonymous, and the pathological picture in both instances is identical. The disease is characterized by thinning of the skeleton with multiple bone cysts, increase of bone osteoclasts, muscular weakness, high urinary calcium excretion and elevation of the blood calcium (Figs. 1 and 2).

THE PERIOTIC CAPSULE

The bony periotic capsule develops, as does bone everywhere, from primitive connective tissue; first into cartilage and later into bone. The cartilage becomes covered externally by a dense connective tissue, the perichondrium, from which is formed the periosteal

Fig. 3. Normal Temporal Bone—Dog. In this section a definite pattern of bone is seen. It is characterized by web-like strands with the bone cells laid irregularly but in a direction more or less longitudinal. The staining is uniform throughout and there is no enlargement of the Haversian canal.

bone similar to that found in the long bones of the skeleton. The cartilage surrounding the membranous labyrinth is displaced by primary endochondral bone in which is imbedded intrachondral bone or cartilage islands, a morphological structure intermediate between bone and cartilage. This primary bone is characterized by its web-like strands with the bone cells laid irregularly but in a direction more or less longitudinal. In long bones around Haversian canals, this primary bone is replaced by lamellar bone. Bast has shown that this primary bone in the capsule persists and Haversian lamella are few in number. In short, the fully formed capsule consists of a persisting primary bone, reticular and not lamellar. Compared with bone elsewhere in the body, it is immature.[9] This statement could explain the response so readily obtained in the bony labyrinth by the administration of the parathyroid hormone, since it has been shown that younger animals react rapidly to hormone administration (Fig. 3).

OTOSCLEROSIS

Otosclerosis is a change in the bone which surrounds the capsule of the labyrinth and which can be definitely recognized histologically as a sharply circumscribed area, usually, but not always, bilaterally symmetrical. In it, cartilage islands so characteristic of the labyrinth capsule are absent. It appears as sharply defined, porous bone. The picture suggests a slow absorption of the primary reticular capsular bone. It has been stated that the typical changes in otosclerosis show that the disease is to be considered a local osteodystrophy, since the details of the alterations are similar to those in von Recklinghausen's disease.[10, 11, 12]

In this experimental work thirty-two animals were used, including guinea pigs, rabbits and dogs. Ten dogs were used, the number of males and females being equal. The time element of hormone administration varied from three weeks up to four months. The most marked bony changes were found, of course, in those animals subjected to the longer period. The amount of A.T. 10 given was determined by the weight of the animal and its individual ability to tolerate the drug. At the same time all of the animals were kept on a synthetic calcium-poor diet, free from vitamin D, since a high vitamin diet has been known to lead to generalized osteoporosis, and the lesion in some way may even simulate that of osteitis fibrosa or hyperparathyroidism. From reports in the literature of experimental osteitis fibrosa, the amount of hormone given was considered sufficient to cause the bony changes hoped for (Fig. 4).

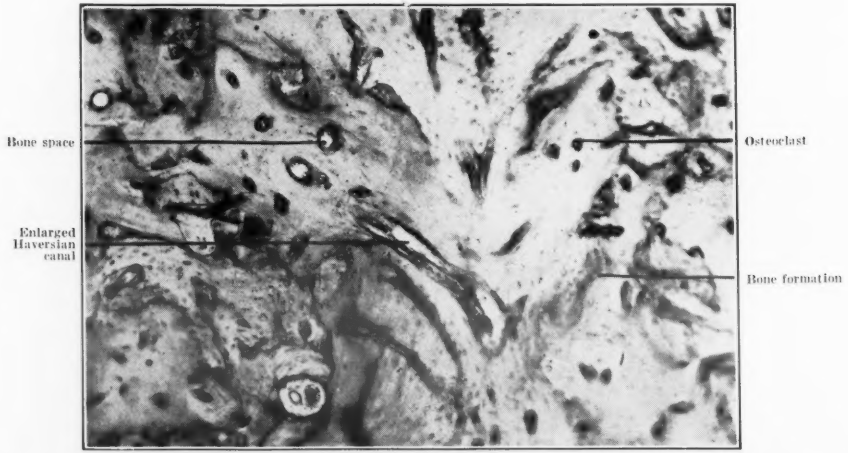


Fig. 4. Experimental Otosclerosis. In the above section the normal mosaic is absent. In certain areas many osteoblasts and osteoclasts are seen. The bone spaces are filled with connective tissue and are of immense size. The section also shows areas of various intensity in staining and marked enlargement of the bone spaces and Haversian canals. The reaction closely resembles that of callus formation following fracture.

FINDINGS

Following the administration of the drug over the desired length of time, in each case the temporal bones were removed in toto and sectioned serially. In turn, each serial section was compared to that of the normal animal. The changes in each animal were bilaterally symmetrical and very little, if any, difference noted pathologically between males and females.

In sections of the decalcified preparations, the conditions found in the different stages of the process showed great variation. In the majority of specimens, however, a sharply circumscribed formation of new bone was observed in the labyrinthine capsule. This bony mass could be differentiated microscopically from the normal osseous tissue by its deeply staining characteristics, by the marked enlargement of the bone spaces and the Haversian canals. The pathological bone is lacking in detail, the normal mosaic being absent. In certain areas marked bony metabolism or formation was evidenced by the presence of numerous osteoblasts and osteoclasts, and without any line of demarcation. The bone spaces are filled with connective tissue and are of immense size, rich in cells and surrounded by numerous large and small blood vessels. The bone corpuscles are more numerous and more compact in the pathologically altered tissue. The reaction closely resembles that of callus formation following fracture in which marked bony metabolism is seen (Fig. 5).

The so-called ankylosis of the stapes was not demonstrated in a single case. It seems reasonable to assume, however, that if the experiment had been carried further perhaps hyperostoses could have been obtained in the region of the oval window and thereby fixation of the stapes. It also seems reasonable to assume from statements made previously that metastatic calcification of the stapes ligament is highly possible. Otosclerosis takes years to develop, while the animals in this series were kept in a state of hypercalcemia for a period of four months at the longest. Therefore, the changes seen in cases of long standing hyperparathyroidism could not be expected.

CONCLUSIONS

1. Hyperparathyroidism is one of the causes of otosclerosis.

2. The lesion of otosclerosis has been produced experimentally.

3. The lesion was produced by the administration of A.T. 10, a parathyroid hormone.

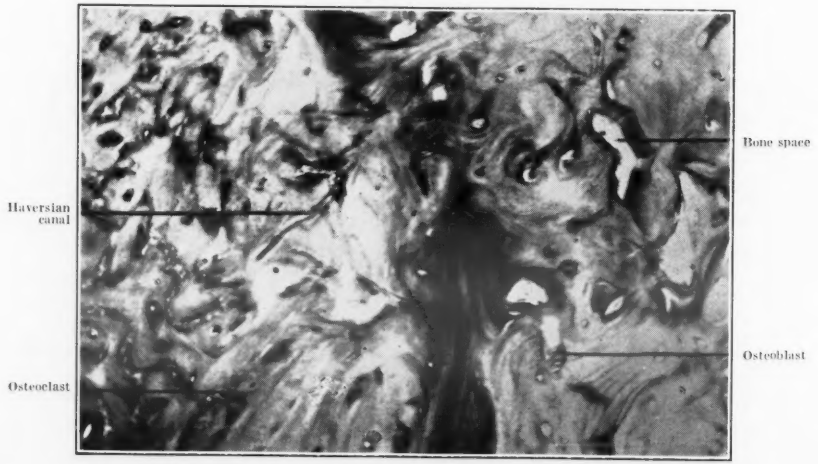


Fig. 5. Otosclerosis. The histological picture seen in the above section is the same as in the experimental sections. The staining varies. Marked bony metabolism is evidenced by the numerous osteoclasts and osteoblasts. There is enlargement of the bone spaces and Haversian canals.

4. The conclusion that osteitis fibrosa represents a healing reaction in bone indicates that it should not be considered a pathological entity.

Slides examined by N. Chandler Foot, M.D., Professor of Surgical Pathology at Cornell University Medical School. Technical work by Mrs. Vivian Hawkins.

243 CHARLES ST.

BIBLIOGRAPHY

1. Cody, Claude C., Jr.: Endocrine Dysfunction to Otolaryngology. Arch. Otolaryng., 30:1 (July), 1939.

2. Cecil, Russell L.: A Textbook of Medicine. W. B. Saunders Co., 1935.

3. Bodansky, Aaron; Blair, John, and Jaffe, Henry L.: Experimental Hyperparathyroidism in Guinea Pigs Leading to Osteitis Fibrosa. J. of Biochem., 87:3, 1930.

4. Alonso, Justo M., and Chiarino, Alberto: Otosclerose ou otospongiose: Influence Exercee par la Parathyroidectomie sur L'Audition. Acta Oto-laryng. (March), 1939.

5. Macleod, J. J. R., and Taylor, N. B.: Observations on the Effects Produced in Normal and Parathyroidectomized Dogs and Herbivorous Animals by Injections of Parathyroid Extract. Trans. R. Soc. Can., 19:27, Sec. V, 1925.

6. Collip, J. B.; Pugsley, L. I.; Selye, H., and Thomson, D. L.: Observations Concerning the Mechanism of Parathyroid Hormone Action. Brit. J. Exp. Path., 15:335, 1934.

7. Best, Charles H., and Taylor, Norman B.: The Physiological Basis of Medical Practice. William Wood & Co., 1937.

8. Bauer, W.; Aub, J. C., and Albright, F.: A Study of the Bone Trabeculae as a Readily Available Reserve Supply of Calcium. J. Exp. Med., 49:145, 1929.

9. Wilson, J. Gordon: Present Status of Otosclerosis. (Personal communication to Dr. H. P. Mosher.)

10. Politzer, Adam: A Textbook of the Diseases of the Ear. Lea Brothers & Co., 1902.

11. Geschickter and Copeland: Tumors of Bone (Revised Edition). Am. J. Cancer, 1936.

12. Shambaugh, Geo. E., Jr.: Chronic Progressive Deafness, Including Otosclerosis. Arch. Otolaryng., 28:780 (Nov.), 1938.

IX

MALIGNANCY OF THE NASAL ACCESSORY SINUSES WITH A REPORT OF TWO CASES OF PRIMARY CARCINOMA OF THE FRONTAL SINUSES

EARLE G. BREEDING, M.D.

WASHINGTON, D. C.

Malignant growths of the nasal accessory sinuses are encountered with moderate frequency, and, although the early literature contains brief reference to the subject, and only few case reports, in the literature of today one finds frequent discussion and records of cases of the various types of neoplasms. It is probable that these tumors existed with some degree of frequency years ago, but the means of diagnosis and treatment have advanced, thus affording earlier recognition of malignancy and a more skillful means of treatment.

The primary carcinoma seems to be the most common variety of malignancy at the present time. Sarcoma, however, is usually seen in younger life and is found most frequently to follow an injury. The maxillary sinus is the one most frequently involved, the ethmoid, sphenoid, and frontal being involved less frequently in the order mentioned, and the involvement is usually secondary, but they may be involved primarily, as reported later in the review of the literature.

The cause of malignant growths of the nose is as little understood as the cause of malignancy in other regions of the body. There is usually some irritant, as chronic sinusitis, pressure from tumors or deformities and abscesses of dental origin. Chronic suppuration is probably the most frequent etiological factor in the carcinomatus growths. In sarcoma there is often a history of an injury to the area involved.

In a recent statistical survey of "Incidence of Malignant Tumors of the Head and Neck", Fabricant[1] found that during the fifth decade of life carcinoma was most frequently seen, and that sarcoma was chiefly found in early life. In other words, malignancy is most commonly encountered in the extremes of life. He also found that these tumors were more common in the male, being seen seven times more frequently than in the female.

Fig. 1. U. S. Army Medical Museum Acc. No. 61214, Neg. No. 68130, is a low power view of a squamous cell carcinoma of the epidermoid type showing fairly well differentiated masses of epithelium infiltrating the stroma and differentiation going to the production of pearls.

"The pathology of these tumors in the nasal accessory sinuses is the same as that of malignant tumors found elsewhere. In carcinoma the infiltrating cells arise either from the surface or glandular epithelium, while in sarcoma the cells are of giant, spindle, or round-cell type" (Barnhill)[2]. As infiltration progresses, and frequently the invasion is slow, the adjacent bone and soft structures become involved, causing ulceration and destruction of tissue. Glandular involvement is late and metastasis is not common according to Thomson.[3]

During the early growth of the tumor, it is usually so insidious that there are no noticeable symptoms. Later, swelling, pain, nasal discharge and frequently bleeding is seen. Symptoms of obstruc-

tion, headache, and foul discharge indicate a further advance in growth. Bleeding may be alarming, due to ulceration involving the nasal blood vessels. After the growth has advanced for some time, anemia, loss of weight, etc., are seen as subjective signs. As previously mentioned, a history of injury may be obtained, and a chronic sinus infection may also be an underlying factor. As a rule, only one side of the nasal passages is involved. When the objective symptoms are very much in evidence the diagnosis is more easily confirmed, especially with the aid of x-ray and biopsy.

Malignancy is often mistaken for sinusitis, dentigerous cysts of the maxillary sinus, fibroma or osteoma. The condition must be differentiated from tuberculosis, syphilis, foreign bodies and rhinoscleroma. Any patient over 40 years of age with a nasal hemorrhage from one side, recurring at frequent intervals, a prominence over the sinus, ulceration, and a fetid discharge, should make one suspicious of malignancy, especially if it is noted that the upper teeth are becoming loose. A hard, immobile growth, rapidly spreading to the adjacent tissue and glands in the presence of a negative Wassermann should be investigated by biopsy.

The prognosis depends upon the type of the neoplasm, cellular structure, age and general physical condition of the patient, the duration and the situation of the tumor. The outlook is not so bad as formerly, and while some cures are reported, and life may be prolonged by appropriate treatment, the ultimate outcome is usually bad.

The treatment depends on the location, the duration of the growth, and the type of tumor. In the cases that are not hopelessly inoperable, internal nasal, external or palliative surgery is indicated. This should be followed by the use of x-ray, radium and diathermy.

Some rhinologists advocate extensive radical surgery, using electrocautery, followed by irradiation. Unfortunately many cases are too far advanced when first seen for appropriate surgery, or, age and the poor general condition of the patient contraindicate surgical intervention. In these cases only palliative treatment is indicated. The pathological report will usually state whether or not the growth is radio-sensitive. If the pain is severe and cannot be relieved otherwise, "divide the 5th or 9th nerve intracranially—Ed",[4] has been advised.

The preceding remarks very briefly cover the general subject of malignancy of all the nasal accessory sinuses. The remarks to fol-

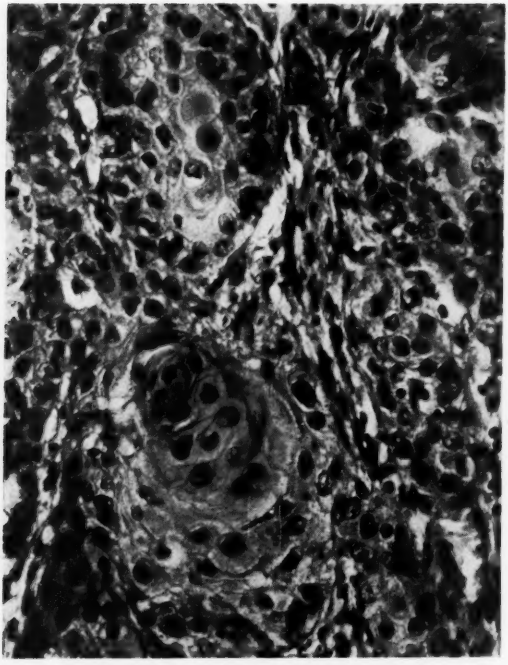


Fig. 2. U. S. Army Medical Museum Acc. No. 61214, Neg. No. 68129, is a high power view of pearls which show the concentric arrangement of the mature cells. The prickles are evident around some of them. There are several splotches of keratin.

low, however, concern malignancy of the frontal sinuses with report of two cases seen by the author during the last year.

Malignancy of the frontal sinus is very rarely seen. While there seems to be some contradiction of the number of cases reported in the literature investigated, I have encountered only 28 cases that are probably authentic. According to Casteran and Achotegin[5], (1937) they found a total of 37 cases in all the world literature, including one case of their own, a primary carcinoma. (This number includes 26 cases reported in 1927 by Portman and Retrouvez.[6]) Their[5] cases included 27 of sarcomata and 10 of epitheliomata. It will be noted in their report that sarcomata predominated nearly

three to one. According to Ringertz[7], "Geschelin[8] in 1934 collected only 19 cases from the literature and added one case of his own, a round-cell carcinoma of the frontal sinus." Ringertz has made, probably, one of the most exhaustive studies that has been found in the literature of the "Pathology of Malignant Tumors Arising in the Nasal and Paranasal Cavities and Maxilla." His report is based on the results of a histopathologic investigation of 391 cases chiefly of malignant tumors in the nasal and paranasal cavities, during the years 1921 to and including 1934. He found that squamous cell carcinoma is the most common form of cancer in the paranasal regions. There were 218 squamous cell carcinomas reported in this author's material. It is of interest to note that this investigator found carcinomata very much more prevalent than sarcomata, as previously reported by Portmann and Retrouvez[6], in France, and Casteran and Achotegin[5] in South America. This has been my impression from the investigation of other reports. For instance, Fabricant[1] reported seven carcinomata and one sarcoma of the accessory sinuses in his tabulation of 249 malignancies of the head and neck.

Referring again to the work of Ringertz[7], there were five cases of malignancies of the frontal sinuses, but this was not histologically verified in all cases.

One of the first cases reported in this country was by Spohn[9] who reported a squamous epithelioma, verified by pathological examination in 1909. In his review of this country, he reported two cases of primary epitheliomata of the frontal sinuses, in 1917. Salinger[11], in his very illuminating article on paranasal sinuses published last year, found reported only three cases of malignancy of the frontal sinus; "Primary Carcinoma Gelatinosum" of the frontal sinus, an extremely rare condition by Garschin. Another malignant tumor of the frontal sinus he records as being reported by Alexandre, the histologic examination of which revealed a hemangiomaendothelioma. The third case he records, reported by Casteran and Achotegin[5], I have already mentioned. The cases of malignancy of the frontal sinus were reported by Spencer and Black[12] in 1938, and Nash[13] in 1935 recorded a round-cell sarcoma of the frontal sinus and orbit in his article on "The Management of Malignancy of the Sinuses." Fabricant[1], whose survey was mentioned previously, found a basal cell carcinoma of the frontal sinus. In addition to the above mentioned reports, I have encountered eleven other cases of malignancies of the frontal sinus reported by: Burger[14] (1932), Claus[15] (1933), Villar[16] (1903), Wisotzki[17] (1913), Brian[18] (1897), Bartha and Onodi[19] (1903), Ruppaner[20] (1917), Schmiegelow[21] (1932), Pie-

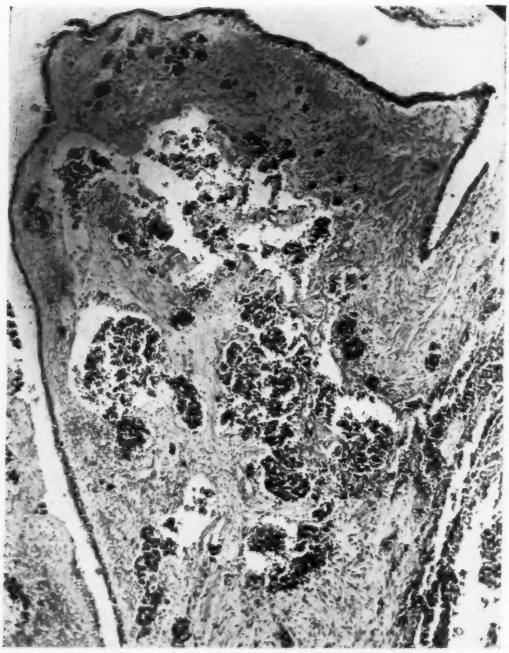


Fig. 3. U. S. Army Medical Museum Acc. No. 59459, Neg. No. 68133, is a low power view of one of the polypoid elements. The stroma is loose from edema, contains scattered masses of mononuclear exudate. The tumor is present as irregular nests of very darkly staining cells which have almost reached the surface in some places. The tumor cells are indistinguishable at this magnification. The epithelium covering the surface of the mass is respiratory in type and can be seen toward the base of the polyp. Much of it has been eroded over the other portions.

trantoni[22] (1927), Iwanoff[23] (1904), and Strazza[24] (1908), complete references to which are found in the bibliography.

Of the twenty-eight cases mentioned in my review, some were undoubtedly included in the report of Casteran and Achotegin[5] in their review in 1937 of thirty-six malignancies and in the nineteen cases reported by Ringertz[7] in 1938. If there were no duplications in these reports a total of only eighty-two cases would be included in this review. Assuming, therefore, that some of the cases were duplicated in the different tabulations, as the bibliography indi-

cates, one must infer that certainly a very small number have been reported to date.

REPORT OF CASES

CASE 1.—Mrs. J., white female, aged 60, was admitted to the Episcopal Hospital June 1, 1938, suffering from headaches, chiefly over the left eye and left frontal sinus, the duration of which was two months. There was severe tenderness over the left frontal area on pressure, and the patient complained of pain in and back of the left eye. Intranasal examination showed congestion of the membranes of both sides and the turbinates were partially covered with a mucopurulent discharge. Tonsils were small but not particularly infected. The larynx was negative. The chest and heart were likewise negative. Blood pressure was 138/80. Patient stated that about a year ago she had an abdominal operation for ulcers of the stomach and was later treated with radium. X-ray examination of the head showed involvement of both frontal sinuses, with pathology in the ethmoid and maxillary as well. Blood examination showed a mild secondary anemia and negative Wassermann. A diagnosis of chronic sinusitis was made, and a bilateral ethmoidectomy with a window in both antra was performed a few days later under local anesthesia. A considerable amount of pus was found in the right ethmoid area. The patient made an uneventful convalescence and was discharged from the hospital 11 days later considerably improved. She was re-admitted to the hospital on my service on July 16, 1938, when I first saw her. She was complaining of recurrence of pain and swelling over the left frontal area. She stated that she had been free from discomfort since her previous admission up to three days ago. There was a marked tenderness over the left frontal area with considerable swelling of the skin, with a mirror-like appearance suggesting an osteitis of this area. The patient's condition showed no improvement under palliative treatment for several days and on July 22nd a bilateral external frontal sinus operation, following the technique described by Beck, was performed. The anterior portion of the left frontal bone was necrotic over the entire area and the posterior portion was eroded through an area of about three-fourths inch in diameter, exposing the dura protruding into the cavity on this, left, side. There was a large mass attached to the necrotic bone and periosteum as well as to the dura, extending well into the right sinus. All diseased tissue was removed and a large opening made into the nasal cavity through the ethmoid areas which had been previously operated and rubber tube drain inserted. A small stab wound was made in the center of the forehead for counter-drainage. Convalescence in the hospital was uneventful and she was discharged on August 5th considerably improved. A specimen was sent to the pathologist whose report follows:

"Tissue from the frontal sinus paraffin section shows a densely infiltrating epithelial structure in which the basal cells are present but in which squamous cells greatly predominate. There is marked keratinization with abundant 'pearl' formation. The cells are quite irregular. Mitotic figures are not common. Diagnosis: Squamous carcinoma, grade II of frontal region 002-881." Signed—J. W. Linsay, Pathologist.

She was seen about four weeks later, complaining of a recurrence of pain. She was referred to the Warwick Clinic where she received thirteen x-ray treatments, after which there was a decided improvement, until December 6th when she was again referred for x-ray therapy due to some recurrence of pain and

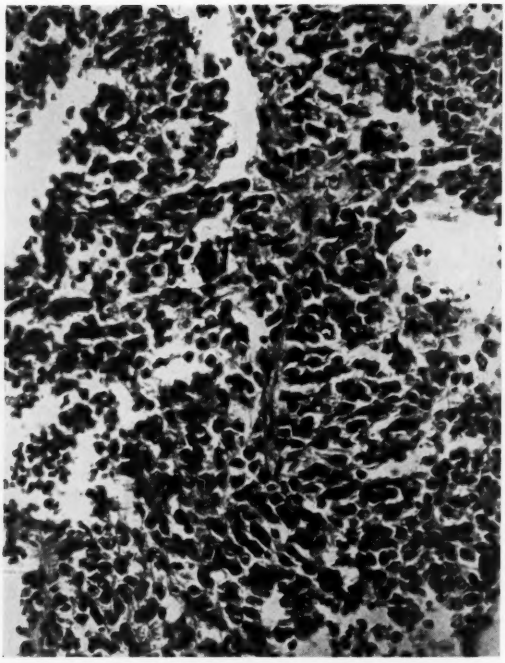


Fig. 4. U. S. Army Medical Museum Acc. No. 59459, Neg. 68134, is a high power view of the tumor showing the transitional type of the cell. The cells are more of the basal type and show no evidence of differentiation toward either the squamous or columnar.

swelling in the frontal area. She was given several weeks' treatment, and a communication from the patient a few weeks ago stated that she was free from pain and quite comfortable, with no evidence of recurrence locally or elsewhere. There will undoubtedly be a return of symptoms in a short time.

CASE 2.—Private male patient, 68 years of age, was referred to me by Dr. Sasscer of Upper Marlboro, Maryland, and was admitted to the Episcopal Hopital August 16, 1938, complaining of pain and swelling over the left frontal area, with apparently a dense, cystic growth protruding from the left fronto-ethmoidal region, resembling a mucocele in appearance. There was nothing of importance in the history of this patient that had any bearing on the slowly progressing growth which he had noticed about two or three months previously. He did, however, state that he had been subject to colds and nasal discharge for many years. The roentgenogram showed "an absorption of superiomedial margin of the left orbit

due to either neoplasm or mucocele; chronic infection with moderate clouding of the right maxillary sinus and the right ethmoidal cells." A left radical external frontal sinus operation was performed August 17th, under general anesthesia. On opening the frontal bone a hard cystic mass filled the entire left frontal cavity extending somewhat into the ethmoidal region. The whole cavity was filled with a necrotic polypoid mass extending into the roof of the orbit and apparently undergoing carcinomatous changes. The growth seemed to have originated within the frontal sinus. The entire frontal and ethmoidal regions were thoroughly cleansed and the maxillary sinus explored, showing only thickened membrane lining the cavity. The wound was closed and intranasal drainage instituted. The specimen was sent to the Army Medical Museum for examination, the histological report of which showed: "The cells are more of the basal type and show no evidence of differentiation toward either the squamous or columnar. This is a more common form of carcinoma arising from respiratory epithelium than the squamous cell." Diagnosis: Transitional cell carcinoma Acc. No. 59489." Signed—Lt. Col. J. E. Ash, Curator.

The patient's stay in the hospital thereafter was uneventful, and he was discharged on the eleventh day, greatly improved. October 15th he was seen complaining of a recurrence of swelling over the frontal area and was referred for x-ray therapy. A communication of January 6th, 1939, from his physician, informed me that he was still receiving x-ray therapy treatments at the Warwick Clinic and was very much improved. Another personal communication from the doctor a few weeks ago stated that metastasis had occurred in the cervical glands and apparently in the lower respiratory areas, and he will, probably, not live longer than a few weeks.*

I believe these two cases had their origin in the frontal sinuses, and notwithstanding the fact that the ethmoid cells showed some involvement in both cases, the gross pathology found at the operations, and histologic study, justify the diagnosis of primary malignancy in the frontal sinuses.

1801 Eye Street, N. W.

BIBLIOGRAPHY

1. Fabricant, N.: Malignant Tumors of the Head and Neck. Arch. Otolaryng., 29:1 (Jan.), 1939.

2. Barnhill, John F.: The Nose, Throat and Ear. D. Appleton & Co., New York, p. 158, 1938.

3. Thomson, Sir St. Clair: Diseases of the Nose and Throat. D. Appleton & Co., New York, fourth edition, p. 307, 1937.

4. The Year Book of the Eye, Ear, Nose and Throat, p. 496, 1938.

5. Casteran and Achotegin: Revista Argentina De Oto-Rino-Larengologia, No. 7-8, 1937.

6. Portmann, G., and Retrouvey: Cancer du Nez et des Fosses Nasales. G. Doin, Paris, 973 pages, 1927.

*The patient died during the latter part of March, 1939.

7. Ringertz, N.: Pathology of Malignant Tumors Arising in the Nasal and Paranasal Cavities and Maxilla. Acta Oto-laryng., Supp. 27, 1938.

8. Geschelin, A. I.: Clinical Aspects of Malignant Neoplasms in Accessory Nasal Sinuses; Primary Carcinoma of Frontal Sinus. Acta-oto-laryng., 21:351, 1934.

9. Spohn, G. W.: Carcinoma of the Frontal Sinus. J. Indiana Med. Assn., 3:402, 1910.

10. Dougherty, D. S.: Primary Epithelioma of the Frontal Sinus: Report of Two Cases. Laryngoscope, 27:37, 1917.

11. Salinger, S.: The Paranasal Sinuses. Arch. Otolaryng., 28:283 (Aug.), 1938.

12. Spencer, F. R., and Black, W. C.: Malignant Diseases of the Nasal Accessory Sinuses, with a Review of Eleven Cases. Laryngoscope, 48:77 (Feb.), 1938.

13. Nash, C. S.: The Management of Malignancies of the Sinuses. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 44:220 (Mar.), 1935.

14. Burger, H., Cancerous Pyopneumatocele of Frontal and Ethmoid Sinuses: Case. Nederl. tijdschr. v. geneesk., 75:5485, 1931; also Acta Oto-laryng., 17:1, 1932.

15. Claus, H.: Difficulties in Differential Diagnosis of Schuller-Christian Syndrome in Carcinoma of Frontal and Ethmoid Sinuses: Case. Ztschr. f. Laryng., Rhin., Otol., 24:391, 1933.

16. Villar, F.: Epithelioma du Point Ayant Perfore L'os Frontal et Envahi la Dure-mere. Assoc. franc. de chir. Proc.-verb. (etc.), Paris, 16:217, 1903.

17. Wisotzki, K.: Ueber das Carcinoma der Stirnhöhle. Deutche Ztschr. f. Chir., 124:605, 1913.

18. Brian: Epithelioma du Sinus Frontal Inoperable. Mem. et Compt.-rend. Soc. d. sc. med. de Lyon (1896), 36:pt. 2, 37, 1897.

19. Bartha, G., and Onodi, A.: Primarer Krebs der Stirnhöhle. Arch. f. Laryngol. u. Rhinol., 15:167, 1903. Also translation, Rev. hebd. de laryngol. (etc.), Paris, 2:553, 1903.

20. Ruppaner, E.: Über primäre Maligne Geschivülste Stirnhöhle. Ztschr. f. Ohrenh. (etc.), 75:365, 1917.

21. Schmiegelow, E.: Primary Cancer in the Frontal Sinus. Hospitalstid., 75: 2, 1932 (Otolaryng. Sect., p. 94).

22. Pietrantoni, L.: Primary Adenocarcinoma of Left Frontal Sinus and Malignant Papilloma of Nasal Septum: Case. Ann di laring., otol., 28:129, 1927.

23. Iwanoff, A.: Ueber Einen Fall von Primärem Carcinom des Sinus Frontalis. Arch. f. laryngol. u. Rhinol., 16:520, 1904.

24. Strazza, F.: Carcinoma Primitivo del Seno Frontale Sinistro. Atti d. Cong. d. Soc. ital. di laringol. (etc.), (1907), Pavia, p. 24, 1908.

X

TUMOR OF THE FACIAL NERVE WITHIN THE MASTOID BONE*

CLAIR M. KOS, M.D.

BOSTON

The specimen to be described was found in a series of approximately 400 mastoid dissections performed in the Postgraduate Course in Otology at the Harvard Medical School. The figures quoted are by no means to be taken as a criterion as to the rarity or frequency of this tumor, but should be regarded as unusual among those cadavers dissected. The information gathered from the literature is disappointing. This tumor, from its gross appearance and relationship to the facial nerve, was suspected of being a neurofibroma. A very lucid description of the histopathology of Schwannoma by Gnassi and Borrone[1] gave a hint as to its probable classification. Recentlv, Williams and Pastore reported a case of neurofibroma of the facial nerve in the facial canal,[2] practically identical in position with the tumor reported here.

The tumor lay in the facial canal in the angle formed by the sigmoid sinus and the posterior wall of the external auditory canal, on a level with the facial nerve. The facial canal was considerably enlarged (Figs. 1, 2, 3).

Naturally, there is no history available and the cadaveric condition of the tissue makes positive histological examination extremely difficult, but the differentiation in the staining quality of the tissue remains the same with the Bodian silver, hematoxylin-eosin, and the Masson trichrome stains.

PATHOLOGICAL EXAMINATION

Gross: Just below the elbow of the facial nerve, as it begins its vertical course, through the mastoid bone, a somewhat fusiform mass, which seemed to be confluent with the nerve, was found. It spread posteriorly in a fan-like manner for a distance of about one cm.; turning anteriorly and superiorly beneath the nerve, and then

*From the Mosher Laboratory of the Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston, Mass.

tapered to an apex. The apex was free from the nerve in contradistinction to the part which was fused with the nerve. The nerve to the stapedius muscle was identified with ease. There were several small filaments from the tumor attached to the adventitia of the sigmoid sinus (Fig. 4).

Microscopic: Microscopic sections showed few cytologic characteristics. The outline of the cells, except in an occasional area, was very difficult to determine. There were numerous cystic areas, the character of which was impossible to make out. Some simulated the structure of veins. The walls appeared to be hyalinized. Hematoxylin-eosin, Masson trichrome and Bodian silver stains revealed bundles and whorls of long, fine, wavy, reticular and collagen fibers surrounding the long axis of the facial nerve. The whole mass appeared to consist mostly of connective tissue, except for the main truck of the nerve, and a few scattered areas which took the nerve stain, but became lost in the substance of the tumor before continuity could be established (Figs. 5, 6, 7).

SUMMARY

The cadaveric condition of the tissue would not permit definite histological study.

Three different stains revealed the same staining qualities of the tissue and they all showed definite characteristics of neurofibroma.

Until the recent publication of Williams and Pastore, there was no record in the literature of a tumor of the facial nerve in the mastoid bone.

CONCLUSIONS

A definite classification could not be given this tumor.

This is the second recorded instance of a tumor of the facial nerve in the mastoid bone.

243 CHARLES ST.

BIBLIOGRAPHY

1. Gnassi, A. M., and Borrone, M.: Schwannoma. Arch. Otolaryng., 27:766-772 (June), 1938.

2. Williams, H. L., and Pastore, P. N.: Neurofibroma of the Facial Nerve in the Facial Canal: Destruction of the Labyrinth and Mastoid Process. Arch. Otolaryng., 29:977-981 (June), 1939.

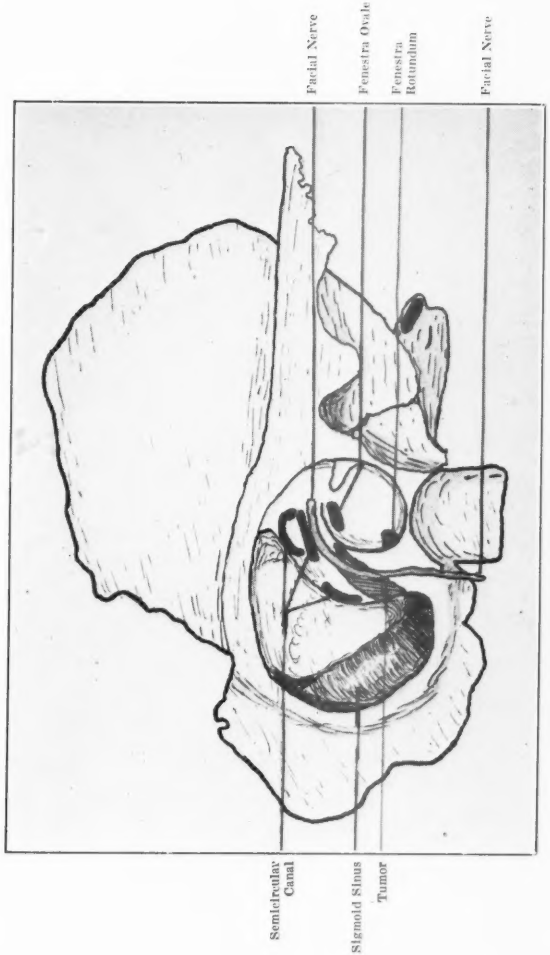


Fig. 1. Semidiagram of the mastoid and middle ear, showing the relation of the tumor to the facial nerve and the sigmoid sinus.

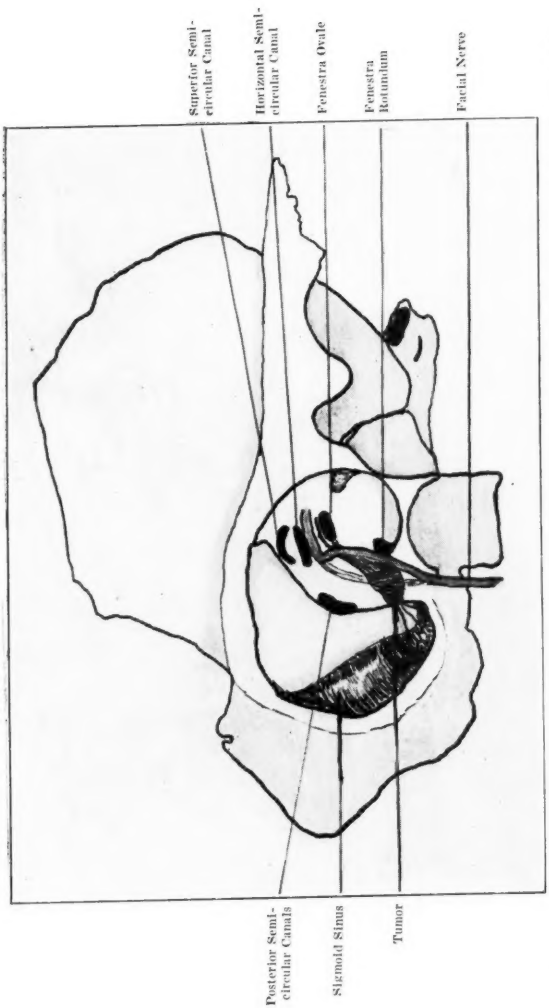


Fig. 2. Semidiagram of the mastoid portion of the temporal bone, showing the tumor displaced anteriorly.

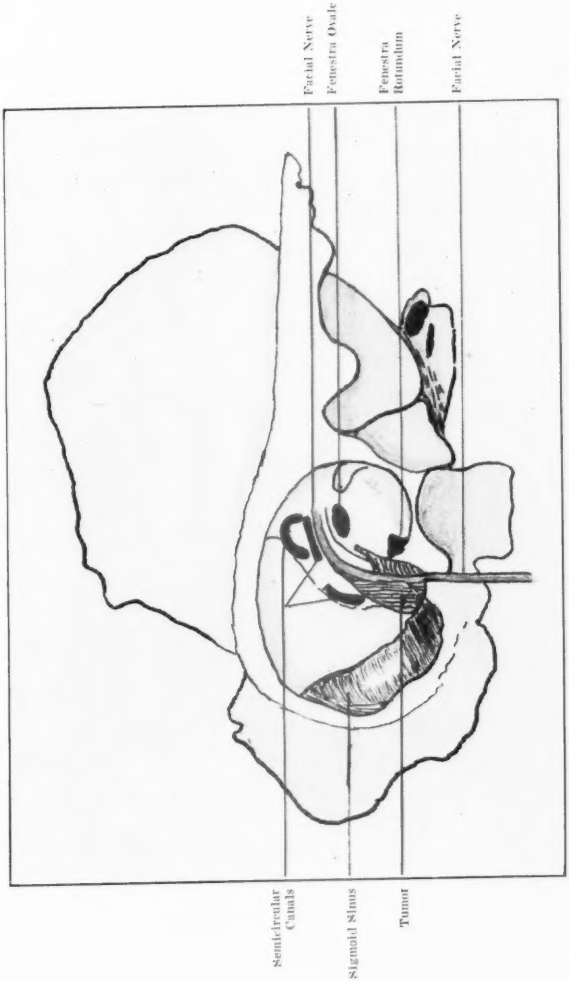


Fig. 3. Semidiagram of the mastoid portion of the temporal bone, showing the tumor displaced posteriorly.

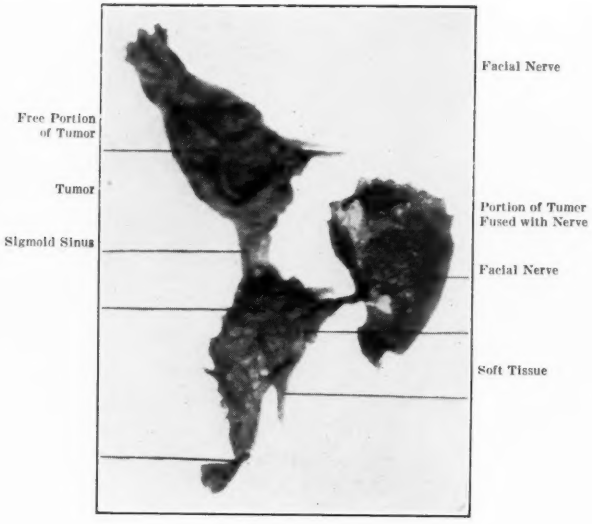


Fig. 4. Enlargement of the original photograph of the resected specimen, showing tumor of the facial nerve and its attachment to the sigmoid sinus.

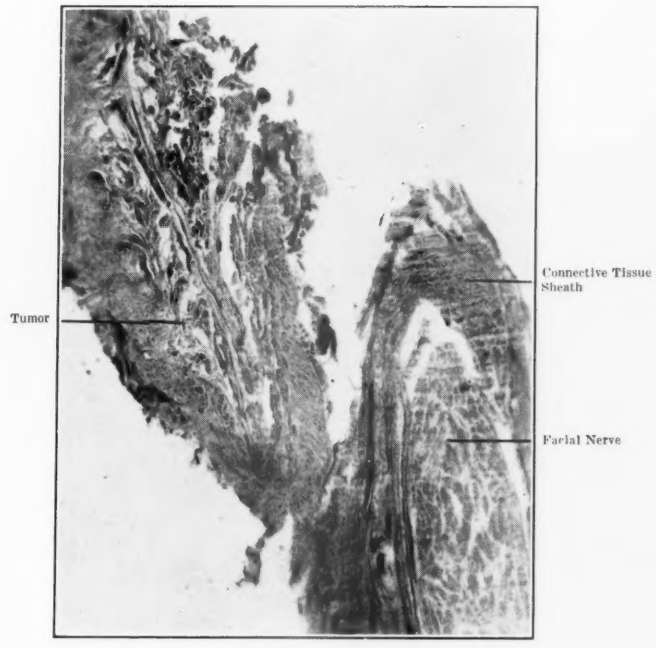


Fig. 5. Low power photomicrograph, showing the facial nerve imprisoned by a thick sheath of interlacing connective tissue fibers. The loosely constructed and predominantly fibrous tumor is confluent with and adjacent to the nerve trunk.

Fig. 6. High power photomicrograph showing cystic character of the tumor. Those delineated with thick fibrous walls suggest the structure of veins.

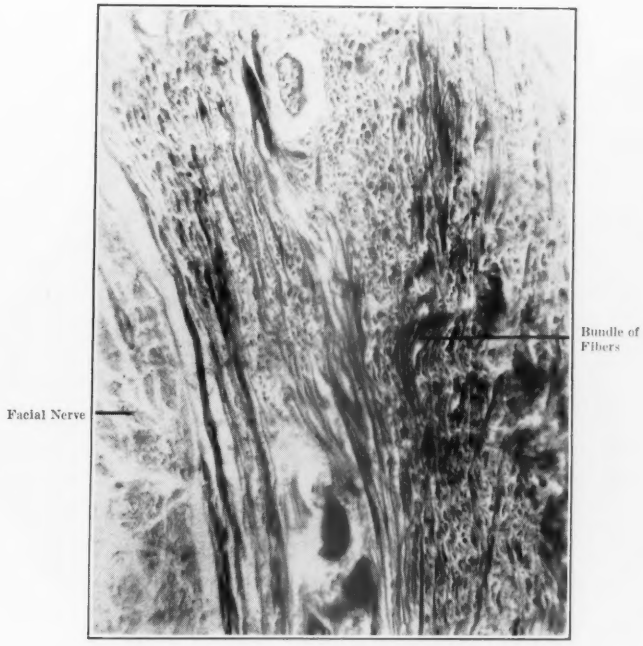


Fig. 7. High power photomicrograph showing eddies and streams of long wavy fibers permeating a matrix of meager cellular definition.

XI

THE PLUMMER-VINSON SYNDROME

David P. Cordray, M.D.

Boston

The syndrome of anemia, glossitis and dysphagia has been evident in medical literature in the past several years. As early as 1914 Plummer noted the association of anemia and dysphagia. Mosher described webs of the esophagus in 1917. In 1922, Vinson reported 69 cases of what he called "hysterical dysphagia." Since this report the syndrome has frequently been referred to as the Plummer-Vinson syndrome. In 1926, A. F. Hurst reported a case in which he was unable to pass the esophagoscope owing to what he termed a "tight spasm of the sphincter." He stated that this case had a "streptococcal glossitis," and an anemia which had preceded the dysphagia by two years. In 1919, A. B. Kelly described the syndrome under the title of "spasm at the entrance of the esophagus." He stressed spasm because of the fact that the folds of the narrowed esophagus gave way without undue pressure and the treatment consisted of dilatation. In the same year, Patterson described a case which showed changes in the mucosa and bands in various positions across the opening of the esophagus, so that the opening was irregular or an obliquely placed slit and not always in the median line. Jones and Owen, in 1928, reported a series of cases in which they described webs and membranes across the lumen of the esophagus and noted that occasionally the esophageal opening was lax and without its usual sphincter-like action. With this as a brief background of the recent thoughts on the subject, we can consider some of the symptoms with particular emphasis on the dysphagia.

SIGNS AND SYMPTOMS

The dysphagia is referred to the level of the larynx and the complaint is made that there is unusual tightness of the throat or there is a feeling as though the larynx were gripping the food. Frequently this symptom is more exaggerated when the individual is tired and is often more troublesome at the evening meal than at breakfast. Often there is a story of having choked on a small piece of meat, bread, or vegetable. In one case reported by Cameron, the choking followed the hasty swallowing of a cocktail in which a

Fig. 1. Normal rat esophagus. Large vacuolated cells in epithelium with no signs of proliferation. Lamellated epithelium not closely packed. No evidence of mitosis.

rather large piece of lemon peel was floating and was caught in the back of the throat. Against this rather sudden onset the dysphagia may be of gradually increasing severity. Sudden attacks may be separated by long intervals during which deglutition proceeds normally. However, the end-result is inevitably the same: the food has to be masticated and swallowed with punctilious care. These patients take a long time at their meals and frequently find themselves more or less ostracized from normal society because of their choking attacks when attempting to eat.

Patterson and Brown Kelly describe the endoscopic appearance of patients who have been affected for years thus: "it was found that the deepest part of the hypopharynx did not present the usual sphincter-like appearance, the entrance to the esophagus appeared as a pinhole or small irregular opening. Sometimes one-half of the mouth of the esophagus seemed closed by a web passing back from

the cricoid." They are therefore of the opinion that the affection is caused by a spasm at the entrance of the esophagus and only exceptionally by a membranous stricture. It is difficult to correlate their endoscopic finding of webs and the conclusions which they have drawn.

The other signs and symptoms associated with this syndrome but not referable to the esophagus are fissures at the corners of the mouth, glossitis, cheilitis, spoon nails and sphenomegaly. Many authors mention this syndrome as occurring during the menopause, and internists have described the dysphagia as hysterical in nature. These have not been discussed because they were not within the scope of our experimental procedure.

BLOOD CHANGES

Because this paper tends to deal primarily with esophageal changes, let it suffice to state that all authors are generally agreed that the anemia is ideopathic hypochromic in character. The main dissenters from this viewpoint are Graham and Johnson who, in 1932, found an increased fragility of the red blood cells and felt that the anemia in these cases was a new and peculiar one of specific type. However, no other authors agree with this viewpoint.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis must embrace all forms of dysphagia at the upper end of the esophagus and after excluding the central lesions which result in bulbar paralysis, the following commoner conditions in this area must be considered: ulcerations from tuberculosis, lues or malignant disease, a stricture following injury or the ingestion of corrosive material, retropharyngeal diverticula, aneurysm, mediastinal tumors and cervical exostoses.

PATHOLOGY

In any attempt to describe the pathology and pathological physiology of this condition, we are faced with the difficulty of determining whether the dysphagia precedes or is the result of the anemia. In some patients, the sign of pallor is present long before the dysphagia becomes apparent. In these it has been presumed that the mucosal changes at the entry of the esophagus are analagous to the glossitis of patients suffering from pernicious anemia and are directly responsible for the dysphagia. However, Cameron suggests that the dysphagia is the first symptom and the dramatic results of treatment suggest that the anemia is a direct by-product of the swallowing

Fig. 2. Pathologic rat esophagus. There is evidence of hyperkeratinization of the epithelial lining. Proliferation of the epithelial elements as evidenced by the closely packed cells near the basement membrane. Increase in the number of mitotic figures. All these changes point toward a precancerous lesion.

difficulty, with the consequent curtailment of food rich in iron and other essentials.

Mosher makes the distinction between bilateral and unilateral webs, holding that bilateral webs are usually acquired and that "other webs, especially if single or behind the cricoid cartilage or just below it, are due to some condition which has given an ulceration of the esophageal mucous membrane." It is unfortunate that many cases of this syndrome were reported by medical men. These men passed bougies blindly and gave no evidence as to whether or not webs were present. Mosher also believes that "many a so-called case of globus hystericus is simply a case of web in the pyriform sinus or behind the cricoid."

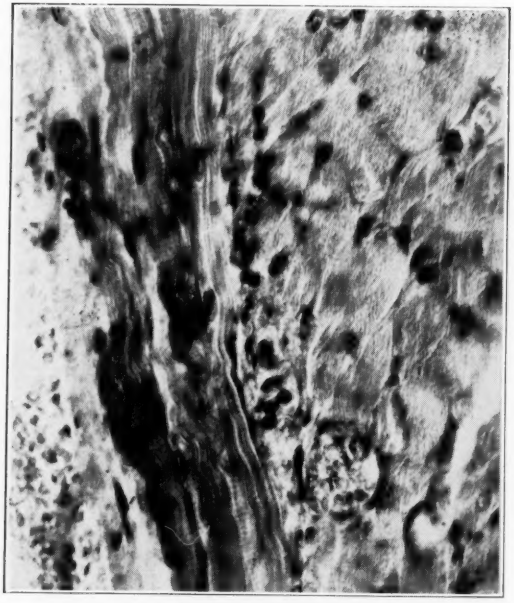


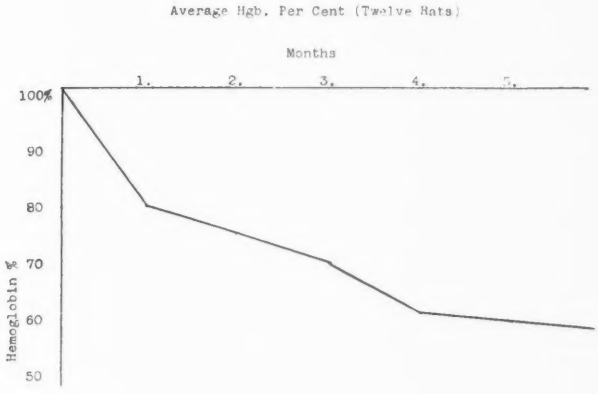
Fig. 3. Musculature of esophagus. Neither the circular nor longitudinal muscle fibers show any change from the normal.

As in many conditions in which the exact etiology is not known, many theories have been advanced to explain the pathology. Hurst of Guys Hospital thinks that perhaps a streptococcus infection in the throat is responsible for both the anemia and the dysphagia. However, agglutination tests for this bacteria in the patient's serum have all proven negative, according to Cameron, and the effects of treatment would appear to negate completely Hurst's suggestion.

Another theory which has never been proven is the so-called neuro-muscular one. The adherents to this theory claim that the dysphagia is the result of a disturbance in the neuro-muscular mechanism which causes relaxation of the normally closed pharyngo-esophageal sphincter, the atrophic condition of the mucous membrane in the neighborhood being the causative factor of this neuro-muscular disorder. Evans (1930) advanced the view that the underlying cause of this syndrome was tertiary lues of the third or fourth generation, but Stokes does not believe this argument.

There are only two autopsy reports in the literature. In view of the number of papers which have been written about this syndrome this is a very singular fact. The first, appearing in an article by McGee and Goodwin, is so brief that little may be learned from it; it simply states "that definite chronic esophagitis with erosion of the epithelial lining was present." The second is from an article by Suzman in 1933, his work having been done at Massachusetts General Hospital, and his comments are as follows: "From the gross findings it is evident that a real obstruction brought about by raised folds of mucous membrane was present. The mucosa and muscle of the tongue and esophagus showed definite histologic abnormalities consisting chiefly of hyperkeratosis of the epithelium with areas of desquamation and of degenerative atrophic changes of the underlying muscle. The presence of areas of mucous membrane simulating leukoplakia and containing immature cells exhibiting mitosis is of interest in view of the tendency of malignant disease to develop."

Because of the wide divergence of opinion concerning the pathology of this condition and the dearth of autopsy material, the following experiment was conducted: Twelve laboratory mice were placed on a diet consisting wholly of milk, the object being to produce a severe anemia. The rats were fed as much of the milk as they cared to drink over a period of five and one-half months. The milk diet contained all of the essential vitamins but was extremely low in iron. The following chart will show how profound was the anemia:



At the end of the five and one-half months period, the rats were weak, definitely underweight and the fur was very coarse and thin. They were sacrificed.

Pathologic sections of all the esophagi were made and a number made of the esophagi of rats (Fig. 1) which had been fed the routine laboratory diet. The microscopic examination of the slides revealed the following: the epithelium of each rat in the group of twelve showed marked hyperkeratinization with proliferation of the cells comprising the epithelial layer (Fig. 2) and increase in the number of mitotic figures over the usual normal number. This points to a precancerous type of lesion. No fibrous tissue replacement in the musculature (Fig. 3) was evident, thus conclusively ruling out that web formation might arise as a result of anemia. Just below the basement membrane, in various sections, were detected epithelial cells which had apparently broken through this membrane. Suzman, Johnson and Ahlbom have written articles indicating their belief that the Plummer-Vinson syndrome is a precancerous lesion, although Suzman is the only one with autopsy material who draws his conclusion by examination of the tissues actually involved.

CONCLUSIONS

1. The experiment points to the fact that the severe secondary anemia in no way predisposed to web formation.

2. In the true Plummer-Vinson syndrome we feel that the web is the cause of the anemia, the web forming a barrier to adequate nutrition.

3. The outstanding pathological change which was noted was the hyperkeratinization of the epithelial lining of the esophagus with proliferation of the epithelial layer and increase in the usual number of mitotic figures. Many pathologists consider this combination as a precancerous lesion. Since the only possible etiologic agent is the secondary anemia, it is reasonable to infer that marked, protracted secondary anemia is capable of producing precancerous lesions in the esophagus.

This study was made under the supervision of Dr. H. P. Mosher in the Throat Laboratory of the Massachusetts Eye and Ear Infirmary. Appreciation is expressed to Mrs. Ralph Hawkins, who helped with the hemoglobin determinations, and to Miss Frances Place, who prepared the sections; also to Dr. Shields Warren of the Palmer Memorial Hospital, who reviewed the slides.

243 CHARLES ST.

BIBLIOGRAPHY

1. Plummer, cited by Hoover, H. B.: The Syndrome of Anemia, Glossitis and Dysphagia. N. E. Jour. of Medicine, 213-394 (Aug.), 1935.

2. Vinson, P. P.: Hysterical Dysphagia. Minn. Med., 5:107 (Feb.), 1922.

3. Hurst, A. F.: Some Disorders of the Esophagus. J. A. M. A., 102:582 (Feb.), 1934.

4. Kelly, A. B.: Spasm at the Entrance of the Esophagus. J. Laryng. and Otol., 34:285 (Aug.), 1919.

5. Patterson, D. C.: Obstruction at the Upper End of the Esophagus. Proc. Royal. Soc. Med. (Sect. Laryng.), 24:1203 (July), 1931, and Jour. Laryng. and Otol., 46:532-35 (Aug.), 1931.

6. Jones, A. M., and Owen, R. D.: Dysphagia Associated with Anemia. Brit. M. J., 1:256 (Feb.), 1928.

7. Cameron, J. A. M.: Dysphagia and Anemia. Quart. J. Med., 22:43 (Oct.), 1928.

8. Patterson, D. C.: Clinical Types of Dysphagia. J. Laryng. and Otol. 34:289, 1919.

9. Graham, G., and Johnson, R. S.: Anemia with Dysphagia. Quart. J. Med., 1:41 (Jan.), 1932.

10. Mosher, H. P.: Webs and Pouches of the Esophagus: Their Diagnosis and Treatment. Surg., Gyn. and Obs., 25:175 (Aug.), 1917.

11. Mosher, H. P.: X-ray Study of Movements of the Tongue, Epiglottis and Hyoid Bone in Swallowing followed by a Discussion of Difficulty in Swallowing Caused by Retropharyngeal Diverticulum, Post-Cricoid Webs, and Exostoses of the Cervical Vertebrae. Laryngoscope, 37:235 (April), 1927.

12. Evans, G.: Notes on Nervous Dysphagia with Special Reference to Its Cause. Practitioner, 125:317 (Aug.), 1930.

13. Stokes, John H.: Modern Clinical Syphilology, 1:734, 1926.

14. McGee, L. C., and Goodwin, T. M.: The Syndrome of Dysphagia and Anemia. Ann. Int. Med., 11:1498 (Feb.), 1938.

15. Suzman, M. M.: Syndrome of Anemia, Glossitis and Dysphagia. Arch. Int. Med., 51:1 (Jan.), 1933.

16. Johnson, L.: Certain Considerations on Dysphagia Associated with Anemia. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 47:809 (Sept.), 1938.

17. Ahlbom, H. E.: Simple Achlorhydric Anemia, Plummer-Vinson Syndrome, and Cancer of Mouth, Pharynx and Esophagus in Women. Brit. M. J., 2:331 (Aug.), 1936.

XII

PATHOLOGY OF THE INNER EAR IN A CASE OF DEAFNESS FROM EPIDEMIC CEREBROSPINAL MENINGITIS

E. W. Hagens, M.D.

Chicago

Because of the scarcity of reports in the American literature on this subject it was believed worth while to record the following case:

Robert B., aged 3, was admitted to the Cook County Hospital January 5, 1937. The history showed that about five weeks previous he developed high fever, projectile vomiting and coma, lasting about one week. On recovering from the attack it was noted that the boy could not hear. About eight days before admission he again developed a high fever. At the hospital the child's condition was found to be serious, the neck rigid and Kernig's, Brudzinski and Babinski signs positive. The impression was relapsing epidemic meningitis. Spinal puncture revealed increased pressure, cloudy fluid, and 850 cells, Pandy 2-plus, and on culture, meningococci. Treatment consisted of supportive measures and antitoxin intravenously. However, the child's condition became worse, death occurring January 23, 1937, or about 46 days after deafness was noted. During the hospital period no otitis media was noted. General postmortem examination showed: suppurative leptomeningitis (epidemic type), focal bronchopneumonia, severe parenchymatous degeneration of myocardium, kidneys and liver, and infectious hyperplasia of the spleen.

The temporal bones were obtained by the author and run through in the usual manner. Grossly there were no signs of infection in either ear.

Microscopic examination of the right ear revealed a number of changes. Horizontal sections at the level of the superior semicircular canal showed an irregular destruction of the bony labyrinth, leaving various sized spaces and channels. Because of this in several sections it was possible to follow a soft tissue pathway from the perilymph of the semicircular canal to the tunica propria of a pneumatic space (Figs. 1 and 2). Both the perilymph and the pathologically formed spaces were filled with connective tissue, containing blood vessels and macrophages. In some sections the perilymph contained a necrotic cellular exudate with occasional polymorphonuclear and multinuclear cells. In the endolymph space were also many cells, some apparently macrophages. There were no osteoclasts in the adjacent bone, nor was there much evidence of bone building. At a

slightly lower level the fossa subarcuate was encountered but without evidence of infection. From an anatomical standpoint pneumatic spaces were found extending laterally around and anterior to the superior semicircular canal (Fig. 1). The crista of the superior canal was indistinct, the cupula being replaced by a mass of necrotic cells. Sections at the level of the cochlea showed extensive vascular connective tissue formation in the scala vestibuli and tympani, especially in the latter in the basal coil (Fig. 3). Large clumps of necrotic cells were present in the scala vestibuli, only an occasional polymorphonuclear being recognized. The organ of Corti was practically absent, its outline appearing only in the apical coil. In places the spiral ligament could not be recognized, this structure being part of the new connective tissue formation. The terminal cochlear and vestibular portions of the eighth nerve were practically absent, only a relatively few poorly preserved fibers being found. A few spiral ganglion cells were noted in the base of the modiolus. In the internal auditory meatus the two divisions of the eighth nerve appeared fairly normal, including Scarpa's ganglion. However, a cellular exudate of macrophages and a number of polymorphonuclears were noted along the nerve sheath. In the vestibule the utricle, saccule and horizontal and posterior cristae were practically unrecognizable, there being an extensive necrotic cellular exudate throughout (Fig. 4). Occasional polymorphonuclears were seen, also a considerable deposit of fibrin in places. The crura of the horizontal and posterior semicircular canals were mostly filled with connective tissue and necrotic cells. The bony edges of the canals were irregularly destroyed, as noted in the superior canal. The ductus and saccus endolymphaticus were filled with connective tissue, with small mononuclear cells and polymorphonuclears also being noted. New bone formation could be seen about the borders of the saccus. Except for trauma at autopsy the stapes and oval window were intact. The fossa for the oval window contained considerable connective tissue and necrotic cells. A similar situation was found in the fossa for the round window. In some sections the pneumatic spaces showed a cellular exudate with thickened tunica propria. The aquaeductus cochlearis was filled with connective tissue containing a number of mononuclears and a few polymorphonuclears.

Horizontal sections of the left ear revealed on the whole a less extensive involvement of the labyrinth. The superior canal was free of pathology and no changes in its bony labyrinth were noted (Fig. 5). This ear also showed extension of pneumatic spaces anterior to the canal (Fig. 5). There were collections of necrotic cells containing occasional recognizable polymorphonuclears in some of the

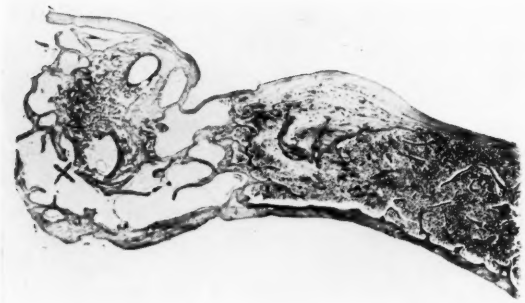


Fig. 1. Right ear, showing extensive pneumatization around superior semicircular canal. At *X* the bone has been eroded.

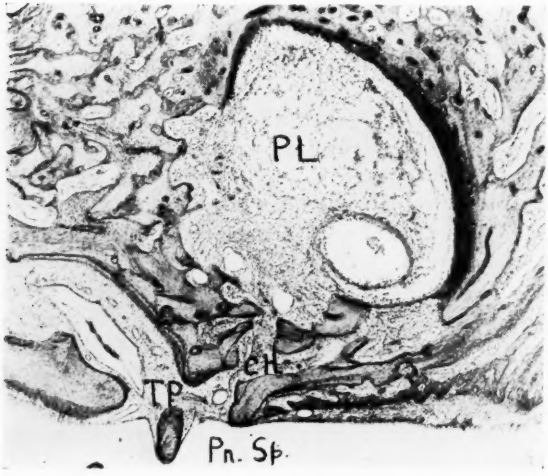


Fig. 2. Right ear. Higher power of the area marked *X* in Fig. 1. The perilymph, *PL*, space is practically filled with cellular material, and as a result bone erosion is in continuity through channels, *CH*, with the tunica propria *TP* of a large pneumatic space, *Pn. Sp.*

pneumatic spaces. The tunica propria was moderately thickened. The fossa subarcuata was free of infection, also the seventh cranial nerve and geniculate ganglion. The crista of both vertical and horizontal canals and the macula acoustica of the utricle and saccule were intact and recognizable; however, the cupulae of the canals and otolith membrane of the utricle and saccule were absent. Only a few necrotic cells were noted overlying the end-organs. Only a little evidence of purulent infection was to be seen in the perilymph or endolymph spaces in the vestibule.

The crura of the horizontal and posterior semicircular canals showed some vascular connective tissue and occasional clumps of necrotic cells. Only slight erosion of the bony canal margins was noted. A cellular exudate containing some polymorphonuclears was found along the eighth nerve in the internal auditory meatus; however, the nerve tissue and Scarpa's ganglion appeared to be normal. In the basal coil of the cochlea the organ of Corti was distorted, although still recognizable (Fig. 6). In some sections cells were present in the scala media. In the rest of the cochlea Corti's organ was absent, both endolymph and perilymph spaces being practically filled with vascular connective tissue. The nerve supply to the cochlea was still intact and the spiral glanglion cells showed only slight shrinkage.

The ductus and saccus endolymphaticus contained connective tissue and a few faded cells. The aquaeductus cochleae was not seen on section but the scala tympani at the start of the cochlea contained connective tissue and necrotic cells. Considerable connective tissue and cellular exudate were found around the stapes at the oval window, and also in the fossa for the round window. However, no evidence of extension to the labyrinth through these avenues was noted.

SUMMARY

A summary of the findings in this case shows that apparently in the beginning there was a diffuse suppuration of the labyrinths. At the time of death, 53 days after the onset, the process had undergone resolution and granulation tissue had filled in considerable of the labyrinth. Bone destruction had occurred about the semicircular canals, but only moderate evidence of bone building was found. In the right ear the spiral ganglion and organ of Corti were practically destroyed, only the faded outline of the end-organ being seen in the apical coil. The vestibular organs in this ear were also gone, although Scarpa's ganglion was intact. In the left ear the spiral ganglion was present, and a distorted organ of Corti seen in the basal coil. The

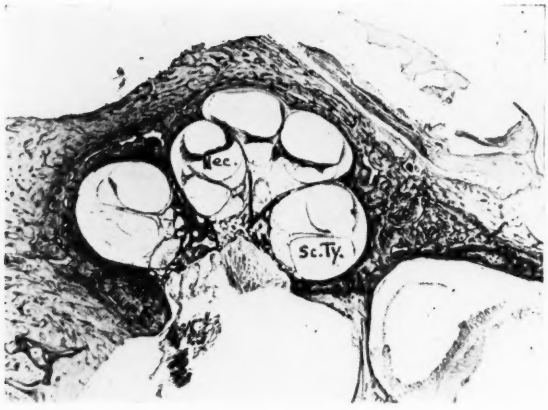


Fig. 3. Right ear. Cochlea, showing extensive granulation tissue formation, especially in the scala tympani, *Sc. Ty.* Necrotic material also present, *Nec.* Organ of Corti atrophied.

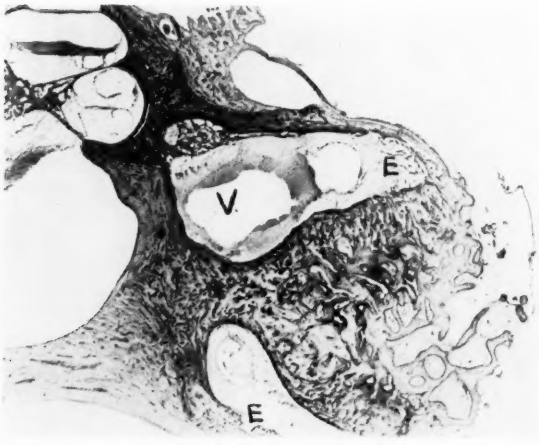


Fig. 4. Right ear, showing extensive necrosis in vestibule, *V.* Erosions, *E,* noted in semicircular canal regions. Cochlea is in left upper corner.

vestibular end-organs were recognized but were no doubt functionless. Both divisions of the eighth nerve were present in the internal auditory meatus of the two ears, but the number of polymorphonuclear leucocytes along the nerve tissue pointed to the fact that the infection spread to the labyrinths along this pathway. The aquaeductus cochlea probably also contributed, as a number of mononuclears and some polymorphonuclears were found here. The fossa for the oval and round windows, and the pneumatic spaces, showed a moderate amount of infection. No pathway of infection from these regions to the labyrinth was noted. The connections from the semicircular canals to the tunica propria of the pneumatic spaces were no doubt due to the bone destruction from the labyrinthitis. The drum membranes and remaining parts of the middle ear were destroyed at autopsy and were therefore not observed.

COMMENT

Politzer[1] presents a valuable discussion of this subject. He states that the deafness arising in the course of the primary form of meningitis becomes evident either immediately after the return of consciousness, between the third and eighth week of the disease, or it develops more or less rapidly during convalescence. Children, as a rule, become totally deaf and show a staggering, uncertain gait, which lasts for some months. The anatomical changes which give rise to disturbances of hearing in this form of disease are: softening or thickening of the ependyma of the fourth ventricle, purulent infiltration and degeneration of the acoustic nerve, embedding of the same in the meningeal exudate, atrophy of the root of the nerve, and purulent inflammation of the membranous labyrinth. The last of these conditions is due to the entrance of the purulent meningeal exudate by way of the aquaeductus cochleae into the labyrinth. The direct action of the bacteria upon the blood vessels of the labyrinth leads to necrosis of the structures of the internal ear, and to inflammatory connective tissue and bone formation, with partial obliteration of the labyrinth cavity. Politzer finds that a vast number of pathological conditions in the labyrinth have been found. In one case both tympanic cavities and vestibules were filled with pus. In another patient a purulent inflammation of the acoustic nerve, saccule, utricle, ampullae and semicircular canals was noted. In a case of cerebrospinal meningitis which had relapsed and in which complete deafness had already taken place during the first attack, the structures of the internal ear had been completely destroyed and replaced by the growth of granulation tissue, a purulent infiltration of the ramus cochlearis and vestibularis, and a filling up of the aquae-

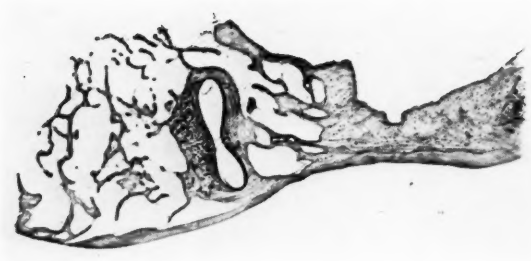


Fig. 5. Left ear. Same level as Fig. 1, to show pneumatic space development, but the lack of infection of the semicircular canal and absence of bone erosion.

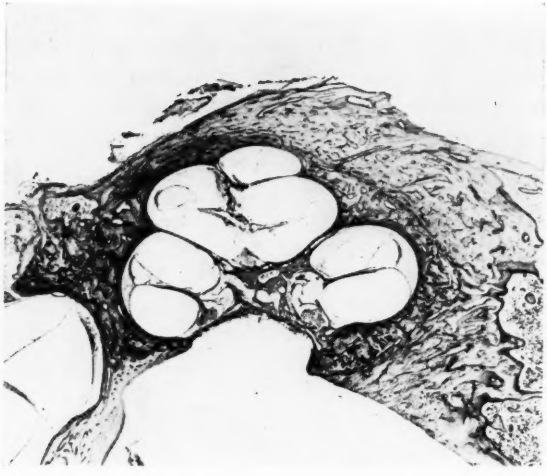


Fig. 6. Left ear, showing cochlea with only rudimentary organ of Corti in the basal coil. Considerable granulation tissue throughout except in the scala tympani of the basal coil.

ductus cochleae with granulation tissue. In another report purulent perineuritis of the acoustic nerve was found, and hemorrhagic and purulent inflammation associated with granulation tissue in the cochlea and vestibule. Politzer emphasizes that from numerous specimens examined postmortem it is known that inflammation of the labyrinth brought about by epidemic cerebrospinal meningitis terminates in the new formation of connective tissue and bone. He quotes Alt, who saw in a case in which the affection had existed fifty-nine days, a new formation of connective tissue and bone nearly filling the entire interior of the labyrinth, in addition to slight purulent infiltration. In another case of sixty-one days' duration, a partial new formation of connective tissue and bone, and a complete destruction of the auditory nerve apparatus was found. Politzer notes that Goerke in 1906 found in 15 cases that the infection of the labyrinth took place by way of the aquaeductus cochleae in three cases, through the aquaeductus vestibuli in one case, and along the auditory nerve in eleven cases. In most of the cases degenerative changes have been observed in the acoustic nerve and in the organ of Corti.

Deafness which remains after epidemic cerebrospinal meningitis is usually bilateral, according to Politzer. When the two ears are unequally affected, the one ear usually becomes totally deaf, while the other loses its hearing power to a marked degree. Disturbances of equilibrium are noted in more than two-thirds of the cases. In a survey made by several of us[2] and reported in 1930, 5,348 children were examined in the schools for the deaf. 2,014, or about 40 per cent of these children, were judged to have acquired their deafness, and of these, 385 gave a history of having developed their deafness following epidemic cerebro-spinal meningitis. 230 were apparently totally deaf, while 155 had some vestiges of hearing. The majority of children, 268 of the 385, exhibited no vestibular reaction. Only 21 cases showed normal responses. These results show that epidemic meningitis tends to produce severe or total deafness with marked or complete loss of the vestibular function.

CONCLUSION

A fatal case of epidemic cerebronspinal meningitis is reported in which deafness occurred 46 days before death and from which the temporal bones were obtained for microscopic examination of the labyrinths. The findings support the reports in the European literature of a severe, bilateral destructive labyrinthitis with a tendency towards healing in the form of granulation tissue and bone. The

end-organs are practically destroyed and demonstrate why permanent total or nearly total loss of cochlear and vestibular function occurs.

30 North Michigan Ave.

BIBLIOGRAPHY

1. Politzer: Diseases of the Ear. Sixth Edition, Lea and Febiger, Philadelphia, 1926.

2. Shambaugh, George E.; Hayden, Daniel B.; Hagens, E. W., and Watkins, R. W.: Statistical Studies of the Children in Public Schools for the Deaf. Arch. Otolaryng., 12:190-245 (Aug.), 1930.

XIII

TUBERCULOUS ULCEROGRANULOMA OF THE TRACHEA AND BRONCHI*

MERVIN C. MYERSON, M.D.

NEW YORK

The subject of tuberculosis of the trachea and bronchi has been receiving increasing attention during the past ten years. The most important single factor in this advance has been bronchoscopy. A new field of investigation has been opened. The pathologist, the clinician, the surgeon, the roentgenologist and the bronchoscopist, acting as a group, have made many contributions. The rapidly increasing bibliography is evidence of the growing interest in this condition. Eloesser,[10] Ornstein and Epstein,[23] Samson,[26] Kernan,[13] and others[1, 5, 9, 11, 12, 14, 17, 24, 28, 29, 30, 31] have added to our knowledge.

Tuberculosis of the trachea and main bronchi has been known to the pathologist for a long time. Autopsy findings[31] have been mentioned for the past hundred years, beginning with the records of Carswell[7] and Louis.[16] The best description of tuberculous bronchitis, especially the form which is the basis of this paper, was given by Loeschke[15] only a few years ago. Tuberculosis of the tracheobronchial tree occurs more frequently than is supposed. In a previous paper,[20] 31 out of 100 tuberculous patients who were bronchoscoped, had evidence of this condition. This study is concerned with 52 cases of the ulcerogranulomatous type of tuberculosis lesion in the trachea and bronchi which were observed among 236 tuberculous individuals who were referred for bronchoscopy during 1938. There were 15 additional lesions in this group which were not of the ulcerogranulomatous type. Each case was referred for a special reason. Statistical data of the incidence of tuberculous lesions in the trachea and bronchi, as revealed by autopsy examinations are available, but such data are unreliable, because many of these lesions represent the terminal stage of the disease.

Ulcerogranulomatous lesions of the trachea and bronchi are more frequently encountered by the bronchoscopist than any other

---

*Presented before the 22nd Annual Meeting of the American Bronchoscopic Society, Rye, N. Y., May 26, 1939.

form of tuberculous disease. This is because they reprensent the active stage of tuberculosis, and they are the most likely to cause signs or symptoms which indicate the necessity for bronchoscopy.

Ulcerogranulomatous tuberculosis implies a lesion of the trachea or bronchus in which the tuberculous process has invaded the wall beyond its surface. This infiltrative process takes the form of granulation tissue. When the superficial layers of this granulation tissue undergo caseous degeneration, there is superimposed upon its surface caseous or necrotic material. This material is white, emulsion-like and fairly adherent. When it dries it becomes flaky and is shed into the lumen. This caseous form of the disease has been called endobronchitis by Loeschke,[15] and caseous necrotic bronchitis by Ornstein and Epstein.[23] It may also be called cariogranulomatous bronchitis. We prefer to speak of it as ulcerogranulomatous bronchitis with caseation. This more clearly defines the local process.

An unusual form of caseous involvement of the bronchus is seen when the entire wall partakes of such a process. When such is the case the lack of nourishment due to a strangled circulation probably accounts for the extensive caseation. (See Figs. 11 and 12.)

At the present time there is no clear conception of the significance of the various manifestations of tuberculosis in the tracheobronchial tree. Tuberculosis behaves in the trachea and bronchus as it does in the larynx. The earliest lesion is the submucosal infiltration, which is recognized by the bronchoscopist as a thickening. This is not a true hyperplasia, as it is frequently called, but rather a localized swelling due to the infiltration of the local tissues by the products of tuberculous inflammation. This infiltrative process may resolve and leave very little or no evidence of its previous existence, or it may progress to penetrate and destroy the mucous membrane and form an ulcer. It may stop at this phase and the ulcer may heal without much evidence of its previous existence. Usually, however, the infiltration in the form of small round cells, epithelioid cells, giant cells and scattered tubercle bacilli, extends beyond the mucosal surface of the involved structure, so that the ulcer is transformed into a granuloma. This process extends downward into the outer structures of the bronchus, just as it extends beyond its surface. Occasionally the ulcerogranulomatous process may be quite superficial. Such a lesion may undergo resolution and healing, so that very little evidence of its former presence exists.

When epithelioid cells predominate, the granuloma does not extend as far beyond the surface as when the lymphocytes prevail. In

the more active and less resistant forms of the disease there is a tendency for the superficial layers to undergo caseation; this has been mentioned before. The fibrotic lesions of the trachea and bronchi constitute the end-result of the ulcero-granulomatous process. These ulcerogranulomatous lesions, therefore, represent the intermediate stage of active tuberculosis of the trachea and bronchi. The fibrosis which follows well established lesions of this type involves all the tissues of the wall, just as the granulomatous process preceding it did. The type and extent of the lesions which are encountered during bronchoscopy depend upon the duration and activity of the disease.

There is no agreement as to the genesis of tuberculosis of the bronchus. Ornstein and Epstein have described a series of cases in which there existed tuberculosis of the trachea or bronchus with little or no manifest disease of the lung. They contend that the frequency of the lesion in the larger bronchi and trachea, with little evidence of pulmonary disease at the onset, and the subsequent invasion of the lung, suggests a different mode of development than the present accepted one of implantation upon the mucous membrane. They believe that in many cases the invasion is from the peribronchial structures, probably from the adjacent lymph nodes, which may have partaken of the first phase of infection, and were not completely healed.

Most investigators feel that the tracheobronchial involvement is directly secondary to that of the lung. Reichle and Frost[25] concluded that the disease spreads to the bronchi from the lung, by way of the lymphatics into the mucous glands. They stress the fact that these glands are found in large numbers between the cartilage and muscles; that lobules of glands are external to the cartilage and lie in close contact with the peripheral alveoli and lymph nodes. They suggest that the lymph nodes may play a part in the infection of the mucous glands. Bugher, Littig and Culp[6] are not in complete agreement with these authors, but admit that bronchial tuberculosis may arise by direct extension from lesions in the lymph glands. In a later paper with Samson[27] they state that the predominant mode of infection appears to be direct contact with tubercle bacilli from pulmonary cavities.

During bronchoscopy, one is strongly impressed by the relatively great frequency with which ulcerogranulomatous lesions ex-

tend from the orifice of a branch bronchus into the main bronchus. Most of these lesions can be traced to a bronchial orifice and their extension can be followed upward from the bronchial orifice.

From this, the observer is led to conclude that there is a definite relation between the bronchial lesion and a caseous pneumonic process in the neighboring lung area. The experiences of Dr. Oscar Auerbach,[2,3] pathologist to the Sea View Hospital, are in accord with this impression. He found involvement of the bronchi close to cavities in over ninety per cent of a large number of cases which he autopsied. There is no doubt, therefore, that these tracheobronchial lesions, with few exceptions, originate from active foci in the lung. These few exceptions are probably explained on a basis of lymphatic extension from the peritracheal or peribronchial region. We have encountered ulcerogranulomatous lesions which are in no way related to a bronchial orifice. The lesions which are unrelated to bronchial orifices are seen especially in the trachea and in the region of the carina.

LOCATION

The location of a lesion which is observed at the time of bronchoscopy usually represents an extension from its point of origin, rather than the actual site of its beginning. Relatively few cases are bronchoscoped early enough to enable the bronchoscopist to see the branch bronchus from which the tuberculous process emerged. The granulomatous process first extends along the wall, which is in the direction of the increased diameter of the lumen, upward toward the larynx. On the right side the process travels along the lateral wall of the main bronchus from the upper lobe orifice, while on the left side the process travels along the superior wall. When the process originates in the lower lobe, the lesion will travel along the posterior mesial or lateral wall, depending upon the location of the branch through which the disease came. When the middle lobe bronchus is the pathway, the disease travels along the anterior wall. Only when the process is fairly early can the path of its spread be visualized. Visualization of the branch of origin is not possible in the later stages, when the granuloma has spread to the adjacent walls, and may have become annular in its distribution.

The location may be confined to the trachea, the main bronchus or a bronchial branch. The granuloma may also be located in the main bronchus and trachea, in the bronchial branch and main bronchus, or in all three, the bronchial branch, the main bronchus and the trachea. Most of the lesions are found in both the bronchial

branch and the main bronchus. The locations of 52 ulcerogranulomatous lesions were as follows:

Trachea	1
Trachea and left bronchus	5
Trachea and right bronchus	3
Right main bronchus	15
Right upper lobe bronchus	7
Right middle lobe bronchus	1
Right lower lobe bronchus	1
Left main and upper lobe bronchus	18
Left lower lobe bronchus	1

A majority of the main bronchial lesions originate from the upper lobe bronchi. This is usually also true of those which extend into the trachea from the bronchi. In three cases the lesion was in the trachea or main bronchus, independent of the branch bronchi. In one of these lesions, which was situated at the carina, the lesion was traceable to the interbronchial glands.

APPEARANCE

The ulcerogranuloma appears as a pale, elevated mass whose surface is rough or irregularly corrugated. It causes varying degrees of stenosis. The lumen of a main bronchus may be completely occluded. Most frequently, however, its diameter is reduced to as little as three or four millimeters. When degeneration of the superficial layers exists, bronchoscopy reveals a whitish, emulsion-like film which is homogeneous and only slightly adherent, so that it can be removed by the suction tip. Occasionally this exudate becomes dry and is encountered in the form of flakes which fill the bronchial lumen and increase the degree of stenosis, at times completely occluding the lumen.

SIGNS AND SYMPTOMS

In hospital practice, the patients are known to be tuberculous when they are bronchoscoped. In private practice, however, patients in whom tuberculosis is not suspected are referred to the bronchoscopist, and bronchoscopy reveals an ulcerogranulomatous lesion. The signs and symptoms can be divided into two groups, those due to the mechanical effects of the lesion and those due to the changes which the lesion itself undergoes.

There is usually a partial stenosis which gives rise to a noisy type of breathing and is described by the patient as wheezing, rat-

tling, snoring, or gurgling. This type of breathing occurs even after the patient has coughed up and expectorated large quantities of sputum. These sounds are frequently heard by others. Some of our patients had been treated for asthma and had had innumerable allergic tests made upon them; in addition they received adrenalin inhalations and injections. These latter have no effect upon this condition. This type of breathing is encountered especially during expiration, but may also occur during inspiration. Not every tuberculous patient who presents a wheeze or noisy type of breathing suffers with this type of disease. We have encountered such breathing, in tuberculous patients, which was due to external pressure by a gland, carcinoma of the bronchus, adherent pulmonary secretions and compression of the trachea or bronchus by mediastinal tuberculous glands.

These granulomatous lesions may act in the same manner as foreign bodies, and their effects are governed by the same laws. They may be partly obstructive, completely obstructive, or may obstruct only during expiration, causing a check-valve arrangement and a resulting obstructive emphysema. Such cases have been reported by Ornstein and Epstein, Clerf[8] and Ballon.[4]

A very striking example of obstructive emphysema due to a caseating ulcerogranulomatous lesion of the left main bronchus was seen in a woman 37 years of age, who was referred for bronchoscopy by a chest specialist. The presence of a bronchial tumor was suspected because there was evidence of obstructive emphysema of the left lung during expiration. Tuberculosis was not suspected. The patient had been suffering with cough and wheezing for several months. The rough chest sound of which she complained was intensified during sleep. Examination disclosed that her left lung was hyper-resonant during expiration. X-ray and fluoroscopic studies revealed an apparently healthy lung during inspiration. During expiration, however, the mediastinum shifted to the right, the left lung remained distended and the right lung lost part of its air content. A diagnosis of ball-valve occlusion of the bronchus was made. Her sputum did not contain tubercle bacilli.

Bronchoscopy revealed fluffy, moist, scaly, white material filling the main bronchus and overlying an ulcerogranulomatous process which appeared to be coming from the left upper lobe bronchus. Smears made from this area revealed an abundance of tubercle bacilli. The diagnosis was ulcerogranulomatous tuberculous bronchitis, with caseation. The bronchial occlusion was eliminated by the bronchoscopy.

Atelectasis of the pulmonary area communicating with the involved bronchus is more likely to occur when caseation is present. Occasionally a granuloma without caseation causes this phenomenon. The caseous material accumulates and acts as an occluding foreign body; at times viscid sputum which attaches itself to the granuloma, does the same thing. Such atelectasis has been seen in two patients whose tuberculous disease was not suspected by the referring chest specialist. The atelectasis may disappear after the patient coughs up the obstructing material, only to recur some time later.

The second group of signs and symptoms are due to the activity of the lesion. The active lesion is responsible for the persistence of a positive sputum in many patients who have had the benefit of apparently successful collapse therapy. Clinicians are now asking for bronchoscopy in such cases, and the basis for their suspicions is usually substantiated.

There is another group of cases where no apparent reason for the spread of disease to a previously healthy area of lung exists. In such cases tubercle bacilli which abound in the superficial layers of the granuloma, may be aspirated into healthy areas of the lung and cause new foci of disease.

The sputum is strongly positive in the presence of an active caseating lesion. It may be negative, however, even when caseation is present. It is frequently more abundant than would be expected from the extent and type of pulmonary disease. The sputum is usually more tenacious than that which is found in pulmonary disease, and varies in quantity from day to day.

Bronchoscopy was requested for the following reasons:

1. As a routine procedure before thoracoplasty.
2. A positive sputum in the presence of an apparently controlled pulmonary condition, as a result of thoracoplasty or pneumothorax.
3. A spread of the disease to healthy lung which was not expected on a basis of the existing pulmonary condition.
4. A wheezing, coarse, noisy type of breathing.
5. Unexplained atelectasis.

The contraindications to bronchoscopy are the same as have been previously outlined. Active laryngeal disease is a contraindication to bronchoscopy. Patients who are acutely ill should not be

bronchoscoped. A recent, profuse hemoptysis is also a contraindication, as is a marked degree of debility.

When the bronchoscopist encounters a granuloma in the lower trachea, or in one of the main bronchi, it is in the interest of the patient's welfare and of good procedure that the bronchoscope should not be forced through these structures so that it presses against the tuberculous lesion. This refers more especially to those processes which are undergoing superficial, caseous necrosis. When this is done there is great danger of dislodging material containing tubercle bacilli and spreading the disease to healthy parts of the lung. For this reason it is advisable to use a bronchoscope of 5-millimeter diameter rather than a 7-millimeter, when such a lesion is suspected Another very good reason for not manipulating these granulomas is the fact that such manipulation may cause a reaction, so that a complete obstruction or check-valve type of obstruction results. For these reasons, it is well not to perform a biopsy on active ulcerogranulomatous lesions. Inactive lesions are recognized by their velvety appearance and by the fact that they are flatter and have a smoother surface. These characteristics indicate that they are progressing toward fibrosis and healing.

In several of our cases which had been previously bronchoscoped, we were able to observe evidence of an advance of the granulomatous lesion in its upper limit, and fibrosis in some other portion. This demonstrated that the ulcerogranulomatous disease previously observed was healing and being replaced by fibrous tissue. In some cases an apparently isolated ulcer was seen a few millimeters above the upper boundary of the granuloma. At a later bronchoscopy the granulomatous process occupied the site of the previously observed ulcer.

Among the 52 bronchoscopic observations of this condition, there were 34 with ulcerogranulomatous lesions only, and 18 in which fibrotic lesions were present in addition. In three of the 52 cases we were able to discern thickening or evidence of submucosal infiltration proximal to the granulomatous process. In five patients bronchoscopy revealed a complete stenosis of a bronchus; in two, the left main bronchus was completely obliterated by a fibrotic closure at its orifice. Because of the displacement of the lower trachea to the right it was impossible to see the former location of the bronchial orifice. The carina also could not be seen. As a result of these changes, the bronchoscope passed through a continuous tube, consisting of the trachea and right bronchus. The lower left tracheal wall and the medial wall of the right main bronchus appeared to be fused

and their point of fusion could not be visualized. In these cases nature supplied her own form of collapse therapy.

TREATMENT

The value of any form of treatment in tuberculous ulcerogranulomatous tracheobronchitis can be seriously questioned. In a previous paper the limitations of bronchoscopy[22] in the treatment of tuberculosis of the trachea and bronchi were stressed. Attempted treatment or instrumental manipulation of these lesions is justified only when the disease is localized and obstructive. When the tuberculoma is well localized and not extensive, electrocautery will be found of value in decreasing its size. It is useless to attempt any form of therapy for extensive lesions of the trachea or bronchi, or both, because in a great majority of cases only the uppermost or proximal portion of the granuloma is accessible; frequently the extent and nature of the process beyond is not easily ascertained, nor is it easily reached for therapeutic attack. Because most of these lesions contain tubercle bacilli in large numbers, there is great danger of spreading the infection to other parts of the lung. Because of the obstruction which these granulomas create, they convert a proper sized bronchoscope into an oversized one.

If the disease is active, the lesion will usually spread during the interval between cauterizations; if it is inactive, neither cautery nor any other treatment is required, for the granuloma is in the process of being replaced by fibrous tissue. Aspiration of occluding caseous material, which causes obstruction, is of value.

As in the case of tuberculosis of the larynx, ultimate healing and recovery depend more upon the ability of the tissues to react favorably and heal than upon the therapy employed.

REPORT OF CASES

CASE 1.—C. W., a white female, aged 27, was referred for bronchoscopy April 14, 1939. She complained of a "snoring" type of breathing which had lasted almost four years. She described this as a rough sound and attributed it to the accumuluation of mucus. Many tests for allergy had been done and found negative. X-ray study revealed a small caseous pneumonic lesion of the right upper lobe and a shadow in the left upper lobe region which was due to a recent spread. Recently her sputum had become positive for tubercle bacilli. She was referred for bronchoscopy because of her noisy breathing and because the spread to her left upper lobe could not be

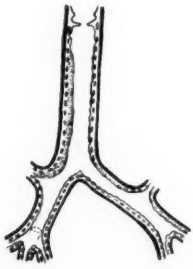


Fig. 1, Case 1. Ulcerogranulomatous process extending from subglottic region, along right tracheal wall and disappearing in the right upper lobe bronchus. All other structures were normal.

accounted for on a basis of the pre-existing pulmonary disease. Bronchoscopy revealed an ulcerogranulomatous process extending from the subglottic region downward along the right lateral wall of the trachea and into the entrance of the right upper lobe bronchus. The carina and all other structures were apparently normal.

Comment: The ulcerogranulomatous lesion began in the right upper lobe and extended from the pulmonary focus to the upper lobe bronchus and upward into the main bronchus and trachea. The extent of the lesion indicated its progress over a relatively long period of time. (See Fig. 1.)

CASE 2.—E. G., a white female, aged 35, complained of cough and expectoration and loss of weight. The onset of her pulmonary disease dated from July, 1935. Her sputum was occasionally positive. In April, 1936, a nephrectomy was performed for tuberculosis of the left kidney. She developed a wheeze in July, 1937. X-ray study shortly after that time revealed a fairly well collapsed left lung due to artificial pneumothorax. Her right lung showed interstitial changes throughout, with fibrosis of the upper lobe. X-ray study in March, 1939, revealed, in addition to the above, a shift of the contents of the mediastinum toward the left. Bronchoscopy was requested because of the wheeze and a suspected atelectasis of the left lower lobe.

Bronchoscopy, November 17, 1937. The entire tracheal wall in its lower five centimeters was the site of a superficial ulcerogranulomatous process. The lower end of the trachea was deviated to

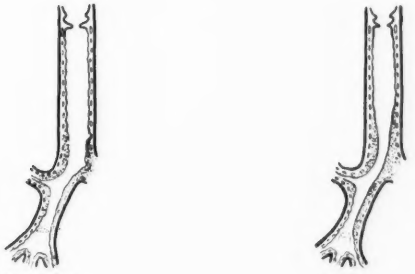


Fig. 2, Case 2. First bronchoscopy: Lower 5 centimeters of tracheal wall and orifice of left main bronchus site of an ulcerogranulomatous process. Orifice completely stenosed. Second bronchoscopy: Stenosis of lower trachea is evident and the left bronchial orifice is replaced by what appears to be a solid wall.

the right. Because of complete stenosis of the left main bronchus, the carina could not be identified. A small, dimple-like depression, with a small amount of granulation tissue, marked the remains of the orifice of the left main bronchus.

Bronchoscopy, April 5, 1939. The lower five centimeters of the trachea presented a markedly stenosed lumen due to flattened, granulomatous tissue. The medial wall of the right main bronchus was affected by the same type of lesion as far down as the middle lobe orifice. The right main bronchus and the trachea made a continuous solid walled tube, because neither the carina nor the left main bronchial orifice could be visualized. The left lower end of the trachea and the medial upper end of the right main bronchus were fused and continuous.

Comment: In this case nature had furnished her own collapse therapy by completely obliterating the passage into the left side of the lung.

CASE 3.—A. R., a white female, aged 53 began her illness with cough and expectoration in 1931. She was admitted to the hospital in May, 1935, complaining of cough and expectoration and pain in the right chest. Her larynx was not involved. Her sputum was occasionally negative. X-ray study revealed effusion in the right chest. There was a diffuse opacity at the right base, with retraction

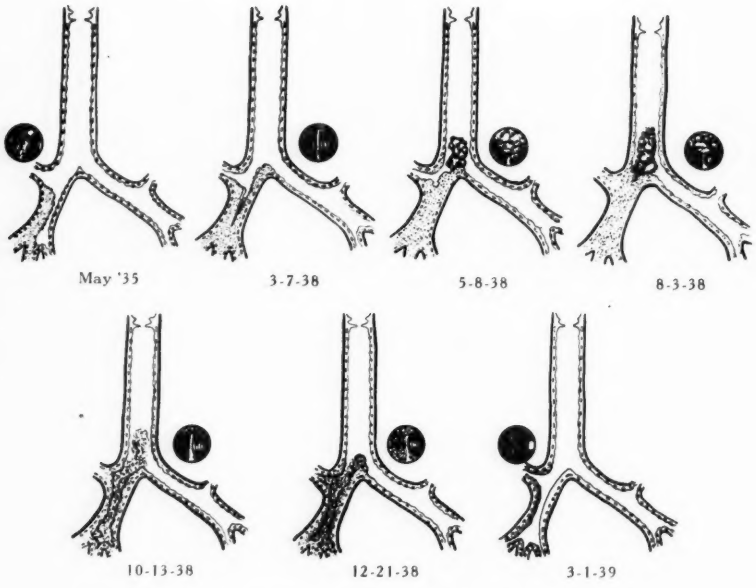


Fig. 3, Case 3. Representation of conditions found at seven successive bronchoscopies.

of the trachea and heart to the same side. An atelectasis of the right lower lobe was suspected.

Bronchoscopy, May, 1935. The right main bronchus, beginning at a point just below the orifice of the upper lobe branch, was found obstructed by an ulcerogranulomatous lesion. It appeared to be more pronounced on the lateral wall.

Bronchoscopy, October, 1937. The previously noted lesion was about the same except that it was closer to the carina on the medial wall.

Bronchoscopy, November and December, 1937. The granulomatous process was flatter, and there was slightly more space in the upper part of the right main bronchus.

Bronchoscopy, March, 1938. Beginning at the carina and extending along the anterior, mesial and posterior walls of the right main bronchus there was an ulcerogranulomatous lesion which extends down to the lower lobe area. The lower lobe orifices could not be seen. The lateral wall of the bronchus was superficially involved

at its beginning for a distance of about two centimeters. The trachea and left main bronchus were normal.

Bronchoscopy, May, 1938. The anterior tracheal wall, beginning about three centimeters above the carina, exhibited an extensive granulomatous process which was partly obstructive at the bifurcation. The most prominent part of the granuloma was cauterized.

Bronchoscopy, August, 1938. The ulcerogranulomatous process in the trachea had extended upward about one centimeter. The right main bronchus was occluded, so that an opening the size of a pinhead remained. Through this opening a purulent secretion discharged, especially during expiration.

Bronchoscopy, October, 1938. The tracheal granuloma previously noted was replaced by a thinly scarred surface. The opening of the right bronchus was no longer the size of a pinhead, but permitted the passage of a 7-millimeter bronchoscope. The anterior wall of the right main bronchus was covered with a caseous exudate. The remainder of the bronchus was red, granular and ulcerated.

Bronchoscopy, December, 1938. A small elevation, covered with whitish material was seen on the anterior tracheal wall, about one and a half centimeters above the carina. The carina was contracted in its antero-posterior diameter, and there was some narrowing of the lumen of the left main bronchus. The right main bronchus had the same appearance as at the previous bronchoscopy and was somewhat narrowed.

Bronchoscopy, March, 1939. The medial wall of the right main bronchus showed evidence of fibrotic change. The lateral wall, especially in its upper part, was still granulomatous. The trachea was free from granulations and had a fibrosed anterior wall.

Bronchoscopy, April, 1939. The medial wall of the right main bronchus presented an extensive ulcerogranulomatous process at a point one centimeter beyond the carina. This occupies two-thirds of the lumen, so that the lower lobe area could not be visualized. The trachea and left bronchial tree were not involved.

Comment: This patient was bronchoscoped twelve times between May, 1935, and April, 1939. The bronchoscopic findings each time gave evidence of the ability of these ulcerogranulomatous lesions both to heal and recur. In this case the disease spread from a focus in the right lower lobe along the bronchial structures and into the trachea. The lesion of the trachea, which was noticed for the first time in May, 1938, healed in October of that year, and remained so.

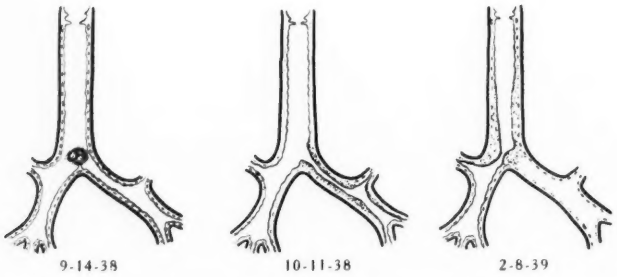


Fig. 4, Case 4. Representation of conditions found at three successive bronchoscopies, showing progress of lesion until the left bronchus was completely occluded and the trachea partly stenosed.

CASE 4.—J. S., a 14-year-old white girl, was found to have tuberculosis of the lung during a routine school study in November, 1937. Her right side was free from disease, while her left lung had recently been collapsed by means of artificial pneumothorax. Her sputum was negative for tubercle bacilli. She was referred for bronchoscopy because of the rough sound of her breathing.

Bronchoscopy, September, 1938. A large, ulcerogranulomatous lesion was seen on the posterior tracheal wall in front of the carina. This spread to the left wall and appeared to extend along the superior wall of the left main bronchus. The carina was thickened and its mucous membrane was markedly congested.

Bronchoscopy, October, 1938. There was a granulomatous alteration of the carinal surface of the left main bronchus. This extended around the bronchial walls to the left upper lobe bronchial orifice. The granulomatous process of the lower trachea was still present.

Bronchoscopy, February, 1939. The middle third of the trachea was the site of a superficial ulcerogranulomatous process which extended almost to the region of the bifurcation. The lumen was narrowed to about 10 millimeters in diameter. The trachea was deviated to the left, and its left lateral wall appeared thickened. The introitus of the left main bronchus was obliterated, while the carina had lost its identity as a result of healing of the previously noted granuloma. The bronchoscope made an oblique passage into the right main bronchus so that the carina and the left bronchus could

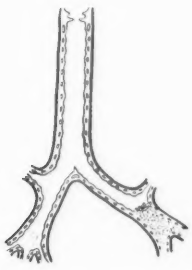


Fig. 5, Case 5. Occlusion of the left lower lobe area by accumulation of caseous flaky material.

not be seen. The structures were replaced by a slight, rounded bulge.

Comment: This case represents the progress of an extensive lesion coming from the left upper lobe and progressing as far as the mid-portion of the trachea. Here, as in Case 2, nature had caused a collapse of the lung by completely obliterating the left bronchial passage (Fig. 4).

CASE 5.—H. R., a woman, 35 years of age, was referred for bronchoscopy by a chest specialist because she had had four episodes of atelectasis of the left lower lobe during the past six months. Two of these were associated with a bronchopneumonic-like process. Her illness had started in June, 1938, with what appeared to be a bronchitis. Two months later she suffered with atelectasis of her left lower lobe, which lasted for two weeks. Her sputum was negative for tubercle bacilli. Her physicians suspected the presence of a foreign body or a neoplasm in her left main bronchus.

Bronchoscopy was performed in December, 1938. The left main bronchus in its lower portion was found occluded by caseous, flaky material. When this was aspirated an ulcerogranulomatous process was seen extending almost as far upward as the upper lobe orifice. Smears made from the flaky material disclosed the presence of tubercle bacilli.

Comment: In this case we encountered an ulcerogranulomatous bronchial lesion, with caseation. The caseous material accumulated and caused obstruction at intervals (Fig. 5).

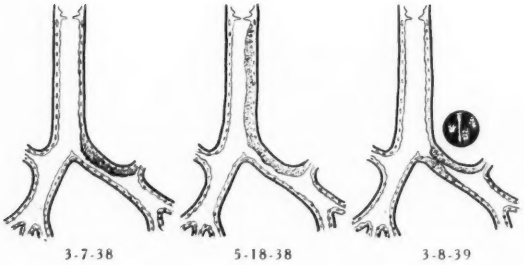


Fig. 6, Case 7. Representation of conditions found at three bronchoscopic examinations. Note that involvement of trachea was no longer present at last bronchoscopy.

CASE 6.—H. S., a woman, aged 28 years, was referred by a chest specialist for bronchoscopy on January 3, 1939. Her illness began in November, 1937. Since then she had had a positive sputum. She had also been given the advantage of pneumothorax, which was 90 per cent effective. Only her right lung was involved. At the time of bronchoscopy there was no x-ray evidence of a cavity.

She was referred because of a persistent cough and a positive sputum in the presence of unilateral caseous pneumonic disease which should have been controlled by the apparently successful collapse.

Bronchoscopy revealed an ulcerogranulomatous lesion of the right main bronchus which extended from the lower lobe area to almost the location of the middle lobe bronchus. This was coated with thin, semifluid, caseous material and reduced the lumen to about six millimeters in diameter.

Comment: The findings at bronchoscopy accounted for the persistent positive sputum in the presence of an apparently successful collapse of a unilateral chest condition.

CASE 7.—A. G., a woman, aged 31 years, was admitted in June, 1936. She was pregnant and complained of cough, expectoration and fever. Her illness began in November, 1935. Left pneumothorax was instituted for the caseous pneumonic disease. There was some exudative productive tuberculosis of the right upper lobe. The sputum was strongly positive for tubercle bacilli.

Bronchoscopy, August, 1937. The left main bronchus was the seat of an extensive ulcerogranulomatous region which appeared to

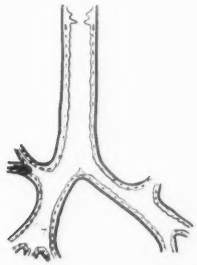


Fig. 7, Case 8. Ulcerogranulomatous lesion occluding axillary branch of the right upper lobe bronchus.

come from the upper lobe orifice. This extended almost to the carina.

Bronchoscopy, November, 1937. Findings in left main bronchus were about the same.

Bronchoscopy, March, 1938. The lesion previously described was now undergoing caseation and superficial layers, so that there was a creamy, white exudate covering it. This was readily removed by the suction tube. The left main bronchia lumen was narrowed to about 10 millimeters in diameter.

Bronchoscopy, May, 1938. The previously noted granuloma now extended up the left side of the trachea to the subglottic region.

Bronchoscopy, March 8, 1939. The trachea was apparently normal, the carina was normal, as was also the right main bronchus. The left bronchus just beyond the carina was occluded by a granulomatous mass covered with whitish, caseous material which appeared to spring from the entire bronchial wall, with the exception of a small section of its posterior portion.

Comment: The bronchoscopies which were performed over a period of eighteen months furnished information as to the extensive spread of the disease from the left upper lobe to the left subglottic region. This was during the period from August, 1937, to May, 1938. The next bronchoscopy after May, 1938, was performed almost a year later and indicated that the tracheal lesion had completely healed, while the left bronchial lesion had progressed (Fig. 6).

CASE 8.—J. B., a man, 35 years of age, was admitted to the hospital in January, 1936. He had been suffering from pulmonary tuberculosis since 1924. He had enjoyed ten years of health when

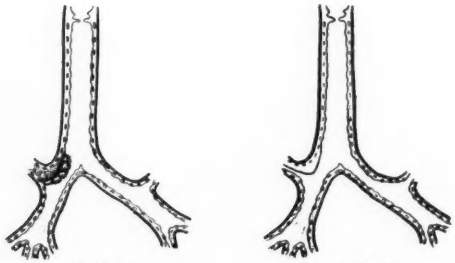


Fig. 8, Case 9. Representation of a granuloma originating in the right upper lobe bronchial area, which was found absent at bronchoscopy one month later.

his trouble recurred. He now complained of occasional cough, blood streaked sputum and loss of weight. The sputum contained tubercle bacilli. The first two stages of a thoracoplasty had been performed in 1936. Because of the persistence of a positive sputum, bronchoscopy was requested before the performance of the third stage of the thoracoplasty.

Bronchoscopy revealed that the position of the right upper lobe bronchus was such as to permit the entrance of a 7-millimeter tube directly into its lumen. A small granuloma was found occluding the axillary branch of this bronchus. (See Fig. 7.)

Comment: This experience demonstrates that the granuloma might be visualized anywhere from its point of origin to the upper trachea if proper means for such visualization are available in the given case. This lesion is unusually small and is a rather early one for the bronchoscopist to encounter.

CASE 9.—I. F., a colored girl, 20 years of age, was admitted in January, 1938. She had complained of cough, expectoration and pain in the right chest for one month. Her sputum was positive for tubercle bacilli. X-ray films revealed that the apical and inner portions of the right upper lobe were atelectatic. Bronchoscopy was requested because of the atelectasis.

Bronchoscopy revealed an ulcerogranulomatous process coming out of the orifice of the right upper lobe bronchus and extending on to the lateral and posterior walls of the right main bronchus. This

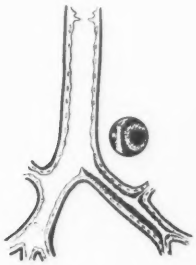


Fig. 9, Case 10. There is an annular involvement of the entire left main bronchus by an ulcerogranulomatous process.

granulomatous lesion was no doubt occluding the apical branch of the right upper lobe.

A second bronchoscopy revealed the absence of the granulomatous process which had been replaced by fibrous tissue and had caused a slight narrowing of the lumen of the right main bronchus.

Comment: An extensive ulcerogranulomatous lesion was encountered at the first bronchoscopy. Thirty-five days later the lesion had healed and was replaced by fibrous tissue. No local treatment was used (Fig. 8).

CASE 10.—R. B., a man, 33 years of age, was admitted in May, 1938. His pulmonary condition was detected in July, 1937. X-ray study revealed an apparently successful artificial pneumothorax on the left side; the right side was uninvolved. Bronchoscopy was requested because of a negative sputum which had become positive despite the fact that the pulmonary disease was not progressing.

Bronchoscopy revealed an ulcerogranulomatous lesion of the left main bronchus which caused a concentric narrowing of this structure from its beginning to the region of the left lower lobe bronchus.

Comment: The endobronchial lesion accounted for the persistence of the positive sputum, despite the apparently successful pneumothorax and the uninvolved right lung (Fig. 9).

CASE 11.—J. A., a 30-year-old man, was admitted in December, 1937. His illness began in 1933. Bronchoscopy was requested to determine the reason for a persistently positive sputum in the presence of a relatively small pulmonary involvement.

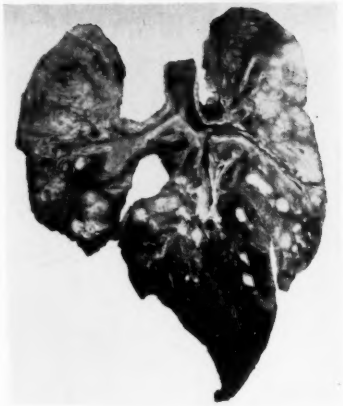


Fig. 10. Autopsy specimen viewed from behind. Left main bronchial wall is greatly thickened and involved in an extensive caseous process, the end result of an ulcerogranulomatous lesion.

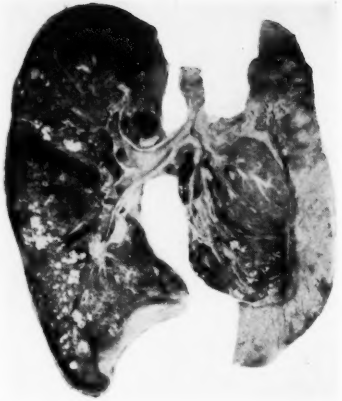


Fig. 11. Autopsy specimen viewed from behind. The lower trachea and right bronchus show a fibrocaseous lesion similar to Fig. 10.

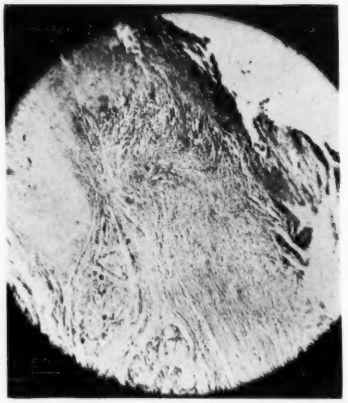


Fig. 12. Photomicrograph of section of wall of right bronchus from Fig. 11. There is an extensive caseation which in certain areas invades and replaces the cartilage. Note also the protrusion of caseous material into the lumen of the bronchus.

Bronchoscopy revealed an obstructing ulcerogranulomatous lesion beginning at the left upper lobe bronchial orifice and extending upward into the left main bronchus, so that only one-fourth of the bronchial lumen remained.

These case reports could be duplicated in the experiences at a large tuberculosis hospital during a single year. They demonstrate that bronchoscopy is of value in the detection of active tracheal and bronchial ulcerogranulomatous lesions whose presence accounts for an unexpected positive sputum, an atelectasis, or an unexpected spread to previously healthy lung.

The ulcerogranulomatous form of tuberculosis of the trachea and bronchus is the most common manifestation of this disease. It represents the intermediate stage of tuberculous infection of the trachea or bronchi. It follows closely upon submucosal infiltration and is transformed into the fibrotic lesion as it heals, as a result of which there occurs a permanent and unyielding stenosis.

Treatment of these lesions is not satisfactory because of their nature and extent.

136 East 64th Street.

BIBLIOGRAPHY

1. Andrews, C. H.: Bronchial Stenosis in Pulmonary Tuberculosis. Can. M. A. J., 33:36, 1935.

2. Auerbach, Oscar: Personal Communication.

3. Auerbach, Oscar: The Pathology of Inflammatory Diseases of the Bronchi. Quart. Bull. Sea View Hosp., 3:134 (Jan.), 1938.

4. Ballon, D. H.: Bronchoscopy in the Diagnosis of Asthma Complicating Pulmonary Tuberculosis. J. Thor. Surg., 5:103, 1935.

5. Barnwell, John; Littig, John, and Culp, J.: Ulcerative Tuberculous Tracheobronchitis. Am. Rev. Tuberc., 36:8 (July), 1937.

6. Bugher, J.; Littig, J., and Culp, J.: Tuberculous Tracheobronchitis: Its Pathogenesis. Am. J. Sc., 193:515 (April), 1937.

7. Carswell, Robert: Pathological Anatomy, London, Logman, 1838.

8. Clerf, Louis H.: Is Bronchoscopy Indicated in Tuberculosis? J. A. M. A., 97:87 (July), 1931.

9. Coryllos, Pol: The Importance of Atelectasis in Pulmonary Tuberculosis. Am. Rev. Tuberc., 28:1, 1933.

10. Eloesser, L.: Bronchial Stenosis in Pulmonary Tuberculosis. Am. Rev. Tuberc., 30:123 (August), 1934.

11. Epstein, H. H., and Ornstein, G. G.: Tuberculosis of the Trachea and Main Bronchi. Quart. Bull. Sea View Hosp., 1:273 (April), 1936.

12. Heaf, F. R. G.: Lesions of the Trachea in Pulmonary Tuberculosis. Lancet, 11:698, 1924.

13. Kernan, J. D.: Bronchoscopy in Pulmonary Tuberculosis. Laryngoscope, 47:777 (Nov.), 1937.

14. Lederer, F. L.: Tuberculosis of the Ear, Nose, Accessory Sinuses, Etc. Goldberg, B: Clinical Tuberculosis. F. A. Davis Co., Philadelphia, 2:37, 1935.

15. Loeschke, H.: Ueber Entwicklung Vernarbung und Reaktivierung der Lungentuberkulose Erwachsener. Beitrage zur Klinik der Tuberkulose, 68:251, 1928.

16. Louis, P. C. A.: Researches on Phthisis, translated by W. H. Walche, London, Sydenham Society, 1844.

17. McConkey, M., and Greenberg, S.: Persistent Rhonchi in the Diagnosis of Bronchial Stenosis Complicating Pulmonary Tuberculosis. Tr. Am. Clin. & Climat. Soc., 50:218, 1934.

18. Myerson, M. C.: Tuberculous Ulceration of the Bronchus. Am. Rev. Tuberc., 19:201, 1929.

19. Myerson, M. C.: Bronchoscopic Observations of Enlarged Tuberculous Bronchial Glands. Arch. Otolaryng., 12:67 (Nov.), 1930.

20. Myerson, M. C.: Bronchoscopy in Tuberculosis. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 43:1139 (Dec.), 1934.

21. Myerson, M. C.: The Value of Bronchoscopy in Pulmonary Tuberculosis. Quart. Bull. Sea View Hosp., 1:261, 1936.

22. Myerson, M. C.: The Limitations of Bronchoscopy in the Treatment of Tracheobronchial Tuberculosis. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 47:722 (Sept.), 1938.

23. Ornstein, G. G., and Epstein, I. G.: Tuberculosis of the Major Bronchi with Little or No Manifest Pulmonary Tuberculosis. Quart. Bull. Sea View Hosp., 1:109 (Jan.), 1938.

24. Phelps, K. A.: Bronchial Obstruction in Chronic Tuberculosis. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 45:1133 (Dec.), 1936.

25. Reichle, H. S., and Frost, T. T.: Tuberculosis of the Major Bronchi. Am. J. Path., 10:651 (Sept.), 1934.

26. Samson, P. C.: Tuberculous Tracheobronchitis: The Role of Bronchoscopy. Am. Rev. Tuberc., 34:671 (Nov.), 1936.

27. Samson, P. C.; Barnwell, J.; Littig, J., and Bugher, J. C.: Tuberculous Tracheobronchitis. J. A. M. A., 108:1850 (May), 1937.

28. Schonwald, P.: Tuberculous Granuloma of the Bronchus. Am. Rev. Tuberc., 18:425 (Oct.), 1928.

29. Vinson, P. P., and Habein, H. C.: Tuberculoma of the Trachea. Surg., Gynec. and Obst., 46:562, 1928.

30. Werner, W. I.: Bronchial Obstruction as a Complication of Pulmonary Tuberculosis Under Artificial Pneumothorax. Am. Rev. Tuberc., 31:44, 1935.

31. Ziegler, E.: A Textbook of Special Pathological Anatomy. Sect. 9-15, p. 786, 1898.

XIV

THE ETIOLOGY AND TREATMENT OF SEVENTH NERVE PARALYSIS*

C. H. McCaskey, M.D.

Indianapolis

In looking back through the literature, we find that the first classical description of facial nerve paralysis by Sir Charles Bell[1] in 1833 could hardly be improved upon today. He said, in his description of a type of left-sided facial paralysis, which has since been termed "Bell's palsy": "The muscles on the left side are wasted and there appears to remain nothing but the thin integuments which hang upon the side of the face, as if dead, without having any action in them, or wrinkles, as in the right cheek; and when he speaks this cheek is alternately puffed out and then collapsed; the air first distending it, as it were a bag, and then escaping at the angle of the mouth. His whole mouth is drawn to the right side, thus producing a most remarkable distortion of the face. Whatever action there is in the mouth is altogether owing to the contraction of the muscles on the right side of it; the left angle hangs loose, and is quite passive; and the saliva is allowed to flow constantly out upon the lower lip on this side."

Bell described many changes of facial paralysis due to different etiological factors, and since that time research and clinical studies have given us a great deal more knowledge as to the etiology and treatment of seventh nerve paralysis. On the basis of our present knowledge, more than one hundred years after Bell's classical description, the following classification of etiological factors entering into facial paralysis may be set forth in order of importance insofar as they affect otolaryngology:

1. Trauma.
2. Infection.
3. Exposure.
4. Neoplasm.

---

*From the Department of Otorhinolaryngology, Indiana University School of Medicine.

5. General systemic disease.
6. Toxicosis.
7. Allergy.

Before entering upon a discussion of these factors, however, it is important to have at least a fundamental knowledge of the anatomy of the nerve concerned in facial paralysis.

The facial nerve consists of two portions, the motor and sensory parts, which emerge at the lower border of the pons in the recess between the olive and the inferior peduncle, the motor part being the more medial. From their superficial attachment to the brain the two roots of the facial nerve pass laterally and forward with the acoustic nerve to the internal acoustic meatus. In the meatus, the motor root lies in a groove on the upper and anterior surface of the acoustic nerve, the sensory root being placed between them.

At the bottom of the meatus the facial nerve enters the facial canal, which it traverses to its termination at the stylomastoid foramen. It is first directed lateralward between the cochlea and vestibule toward the medial wall of the tympanic cavity; it then bends suddenly backward and arches downward behind the tympanic cavity to the stylomastoid foramen. The point where it changes its course is called the geniculum, it presents a reddish ganglion, the geniculate ganglion. Of importance are the *iter chordae posticus* and *iter chordae anticus*, because they are the points where the chorda tympani enters and emerges from the tympanic cavity. After passing through the styloid foramen the nerve divides into the main branches of distribution. The branches of the facial nerve are of two types: the branches of communication, and the branches of distribution. They are as follows: In the internal acoustic meatus, a branch of the acoustic nerve goes to the geniculate ganglion, and the following communications are sent out: One goes to the sphenopalatine ganglion through the great superficial petrosal nerve; one to the otic ganglion by the way of the lesser superficial petrosal nerve; and a branch to the sympathetic plexus of the middle meningeal artery. In the facial canal a branch is sent to the auricular branch of the vagus. At its exit from the stylomastoid foramen, branches communicate with the vagus, the great auricular, and with the auriculo-temporal. Behind the ear a branch communicates with the lesser occipital. On the face a branch communicates with the trigeminal.

The branches of distribution within the facial canal are as follows: A branch is given off supplying the stapedius muscle, and

there the chorda tympani leaves the facial nerve proper. At its exit at the stylomastoid foramen, branches are given off to the posterior auricular, the digastric and styloid muscles, and on the face the branches are temporal, zygomatic, buccal, mandibular and the cervical, supplying chiefly the muscles of expression.

In a division of the nerve into parts for the purpose of studying the etiological factors which cause seventh nerve paralysis, the nerve may be divided at the nucleus into two general divisions, the supranuclear division and the infranuclear division. This was formerly thought to be sufficient, as the symptoms due to lesions of these two divisions are quite distinct; it is possible, however, to further divide the infranuclear division of the nerve into three subdivisions as an additional aid in diagnosis. The first subdivision may be termed the nucleo-geniculate, as it extends from the nucleus to the geniculate ganglion; the geniculo-foraminal region then becomes the second subdivision (i. e., the region from the geniculate ganglion to the stylomastoid foramen); and the terminal portion of the nerve from the stylomastoid foramen to the terminus represents the third subdivision. Let us review these divisions and subdivisions once more: the nerve is first divided into two main divisions, the supranuclear and the infranuclear; the infranuclear division is then further divided into three subdivisions, the nucleo-geniculate, the geniculo-foraminal, and the terminal.

Now that we have the anatomy and the various divisions of the nerve well in mind, we may proceed to a discussion of the etiological factors previously set forth, namely, trauma, infection, exposure, neoplasm, general systemic disease, toxicosis, and allergy.

Trauma presents the one etiological factor which is of prime importance to us. Trauma may be due either to external violence, or to surgery. In the case of the former any division may be involved.

The supranuclear division may be involved by injury to the skull, by producing a contusion, laceration, edema or hematoma.

Fractures at the base of the skull may involve the nucleus of the nerve, and in this event there may be bilateral paralysis, or there may be only unilateral paralysis. Very often there may be an involvement of the other cranial nerves whose nuclei lie in the same region.

The nucleo-geniculate subdivision may be involved in fractures at the base of the skull. In this condition there is complete peripheral paralysis of the nerve, but the chorda tympani is not involved.

Fractures at the base of the skull, involving the geniculo-foraminal subdivision of the nerve occur in the area occupied by the facial canal in the petrous portion of the temporal bone, and cause a complete peripheral paralysis and a disturbance in taste in the anterior two-thirds of the tongue, thereby showing an involvement of the chorda tympani nerve.

There may be paralysis due to fracture through the facial canal very low down, affecting the terminal subdivision, but not involving the chorda tympani nerve. In the terminal subdivision of the nerve, lacerating, gunshot, or stab wounds may involve the main terminal branch or either of the branches of the main terminal subdivision. These cause complete paralysis or paralysis of the muscles supplied by the branches of the terminal subdivision.

Trauma due to surgery is the one etiological factor with which the otolaryngologist is most deeply concerned. This occurs practically always in the geniculo-foraminal subdivision of the nerve as it courses the facial canal, although surgical trauma may occur outside this subdivision of the nerve, as in mastoid surgery of infants or very young children when the incision is carried too far forward, involving the terminal subdivision of the nerve.

The nucleo-geniculate subdivision of the nerve may be involved in surgery of the auditory nerve, surgery of the petrous apex, or in surgery of the gasserian ganglion. In intentional trauma due to resection of the terminal portion of the nerve to relieve chronic clonic spasm, the terminal subdivision is involved.

In doing mastoid surgery, especially in a radical mastoidectomy, the nerve may be severed, or partially so, and the point where this is most likely to occur is where the nerve arches downward behind the tympanic cavity in the region of the *aditus ad antrum* just below the horizontal semicircular canal, or it may occur in the facial canal below this area. Should the canal course through the mastoid cavity in an anomalous position, and this severance occur, there may be a complete paralysis of the nerve, as well as of the chorda tympani nerve.

In doing surgery of the mastoid, trauma to the facial canal may cause paralysis by causing impingement of the bony canal on this division of the nerve, thereby causing a permanent or temporary paralysis of the nerve.

In dressings and drainage which are used in the mastoid cavity paralysis may occur in the nucleo-foraminal portion of the nerve if

the nerve has been exposed by surgery, or there exists a dehiscence in the bony canal.

Infection, although of secondary importance, is responsible for many cases of facial paralysis, and the location of the infection has to do with the division of the nerve involved.

The supranuclear portion may be involved in brain abscess. This might well be classified along with neoplasms, but, after all, it is due to an infectious process and will be classified as such. Whether the paralysis is due to intracranial pressure or due to destruction of the pathway of the seventh nerve to the cortex, the symptomatology produced is the same, paralysis of the lower portion of the face.

Meningitis, or encephalitis may involve this division of the nerve, but they are more likely to involve the nucleus or nucleogeniculate subdivision of the nerve.

Geniculitis or *herpes oticus* is thought to be the direct, or, perhaps, the end-result of infection. This has been demonstrated clinically in influenza. Facial paralysis, usually a secondary complication of this condition, comes on after the herpes appears. We digress from the subject matter at this point to mention the sensory portion of the seventh nerve. We may conclude from the pain manifested in this condition that the seventh nerve must carry sensory fibres as well as gustatory and motor fibres. These fibres appear to be distributed from the geniculate ganglion and are evidenced by postauricular pain, pain in the external portion of the external auditory meatus and pain about the anterior nares and the soft palate. A discussion of this phase of the seventh nerve was brought out by J. Ramsay Hunt[6, 7] in 1907, and at a later date was further corroborated by his findings and those of others.

Acute suppurative otitis media is responsible for many cases of paralysis of the seventh nerve and involves the geniculo-foraminal subdivision of the nerve which lies in close proximity to the middle ear, and is caused either by periostitis or inflammatory change in the nerve itself, producing a pressure paralysis and involving, at the same time, the chorda tympani nerve as evidenced by loss of taste in the anterior two-thirds of the tongue.

In acute surgical mastoiditis there is at times a paralysis involving the geniculo-foraminal portion of the nerve. This may be caused by a severance of the nerve in an anomalously placed facial canal in the mastoid cavity, or by an impingement on the nerve by the wall

of the facial canal due to trauma of the canal wall, or by producing an inflammatory reaction in the canal wall. As we observed in one case, the surgeon, in attempting to locate the antrum, had gone through the anterior wall of the mastoid cavity and obliterated the facial canal and destroyed the nerve at this point.

Chronic suppurative otitis media is responsible for many cases of seventh nerve paralysis and usually involves the geniculo-foraminal subdivision of the nerve. This is due either to necrosis of the bony canal at the point where it courses along the upper internal wall of the middle ear cavity or from pressure absorption by complicating cholesteatoma.

Exposure, better known as the cold air palsy of Bell, occupies third place in our observation of these cases, and occurs in persons who have one side of the face exposed to a chilling draft over a considerable period of time, producing an inflammatory change in the geniculo-foraminal subdivision close to its exit from the stylomastoid foramen either in the canal or in the terminal subdivision just after its exit through the stylomastoid foramen.

Neoplasm may involve any division of the nerve, and it may be benign or malignant.

Any tumor involving the pyramidal tract involves the supranuclear division of the nerve.

The nuclear portion of the nerve may be involved by cerebellar-pontine angle tumors. This may produce unilateral or bilateral paralysis as well as involve the nuclei of neighboring nerves, especially the sixth and the acoustic nerves. Acoustic nerve tumors, as well as cerebellar-pontine angle tumors, may involve the nucleogeniculate subdivision. Exostoses of the internal auditory and facial canal may produce paralysis of the geniculo-foraminal subdivision of the nerve.

Intratemporal epidermoids as reported by Jefferson[8] may produce progressive seventh nerve paralysis involving the geniculo-foraminal subdivision of the nerve.

Malignancies of the middle ear and mastoid cause paralysis by involving the geniculo-foraminal and the terminal subdivisions of the nerve.

Tumors arising between the angle of the mandibular process and the mastoid process and tumors of the parotid gland involve the terminal subdivision of the nerve.

General systemic disease seems to play a part in seventh nerve paralysis, and in this condition any division of the nerve may be involved. This has been noted especially in gout, diabetes, syphilis, tuberculosis, leukemia, poliomyelitis and cardio-vascular disease.

Toxicosis enters into the picture of seventh nerve paralysis, but only in a minor way, and this only when there has been a very severe attack of diphtheria, or when alcohol has been indulged in to excess; also some drugs have been known to produce this type of paralysis. Paralysis due to toxicosis may involve any division of the nerve.

Allergy can, we may be sure, produce paralysis of the seventh nerve. Nothing can be found in the literature to support this. However, I have observed a case of a male, acutely allergic to milk, who manifested this condition. After being off milk, or any of the dairy products for three days, he recovered from the paralysis. In this case, the geniculo-foraminal subdivision of the nerve was involved, as the symptoms clearly showed, since the chorda tympani nerve was also involved.

This concludes the discussion of the etiological factors involved in seventh nerve paralysis. To summarize briefly, the supranuclear division may be affected by trauma, infection, neoplasm, general systemic disease, or toxicosis. In the infranuclear division, the nucleo-geniculate subdivision may be affected by trauma, infection, neoplasm, general systemic disease, or toxicosis; the geniculo-foraminal subdivisions may be affected by any of these or by exposure, or allergy, and the terminal subdivision may be affected by trauma, exposure, neoplasm, general systemic disease, or toxicosis.

Having noted the etiological factors responsible for paralysis in the various divisions of the nerve, we may turn our attention to the treatment of this condition, which will be discussed under two headings, i. e., non-surgical and surgical.

The non-surgical treatment of seventh nerve paralysis consists of medical treatment, physical therapy, and manipulative treatment.

The medical treatment would seem to be best suited to those cases which are due to general systemic disease, and would be confined to such drugs as are suitable in relieving the general condition which is responsible for paralysis.

Physical therapy would be confined to such procedures as application of heat and electrical currents, especially the galvanic and faradic currents. Of course, it is a question as to how much thera-

peutic value these have and, as one writer has said, "It seems the greatest value we obtain from this sort of procedure is that we may by such means keep our patients under observation a much greater length of time, and this enables one to make a more comprehensive study of the case." This, in view of the fact that a great many cases eventually clear up without other treatment, is worthwhile, as these patients are prone to become quickly discouraged and are usually the prey of various cults and unethical practitioners.

Manipulative treatment is a form of non-surgical therapeutic treatment which seems of some importance in these cases, especially if the paralysis disappears at a later date. This has a tendency to keep the muscular tone intact, and when the nerve has returned to its function, much has been gained. It may be applied in the way of massage either by hand or by vibratory methods plus the voluntary effort on the part of the patient to produce movement of the muscles.

Surgery to relieve seventh nerve paralysis may be divided as follows: intracranial, plastic, anastomosis of the seventh nerve with other motor nerves, repair of the seventh nerve proper, and decompression.

We will not discuss intracranial surgery other than to say that this is necessary in the treatment of tumors, hematoma, cerebellar abscesses, etc., and should be delegated to the field of the neurological surgeon.

Plastic surgery should also go to a specialist in that field. It may be used to give facial muscles more tone and support and should be attempted only by the plastic surgeon.

In correcting the paralysis of the seventh nerve by anastomosing the terminal subdivision to the central portion with some other motor nerve, two nerves have been used in the past; namely, the spinal accessory and the hypoglossal. While such anastomoses have advantages, the disadvantages must be considered.

An anastomosis with either nerve gives tone to facial muscles, and better facial expression. The disadvantages are summed up best by Kerrison,[9] who stated the following: "Perhaps nerve anastomosis as to a divided facial nerve failed of facial results because it outraged some law of nature. Such a hypothetical law may be stated as follows: Various motor nerves may supply potential motor power to the peripheral segment of any divided motor nerve; but to expect them to supply such extraneous motivation without simultaneous

excitation of muscle groups which their central nuclei were designed solely to motivate or control, is to count upon the abnormal and, therefore, upon the improbable."

If the patient is prone to accept the seventh nerve paralysis with good grace, it would be much better for him to retain it than to produce a dysfunction of some other nerve, but should the patient be suffering mental anguish, which the paralysis may bring about, one would best try to correct the defect by making an anastomosis with one or the other of these nerves, preferably the hypoglossal.

In clonic facial spasm it is necessary, if we are to relieve the condition, to resect the terminal subdivision of the seventh nerve and thereby produce a facial paralysis. A repair of the paralysis so produced may be accomplished by anastomosing either the spinal accessory nerve or the hypoglossal nerve. A complete discussion of this condition is presented by Gilbert Phillips[10] of the University of Sydney, and in a paper by Charles Coleman,[3] Medical College of Virginia.

A final method of correction of the seventh nerve paralysis is the end to end anastomosis of the severed nerve, as the nerve is often accidentally severed in doing mastoid surgery.

Those cases which are due to accidental injury to the geniculo-foraminal subdivision of the nerve in doing mastoid surgery, those following acute infections of the middle ear, and those cases which are due to exposure and are classified as the cold air palsy of Bell are best suited for surgery and are the ones which are most likely to have the best results. It was not until Ballance and Duel conducted their research on the operative treatment of facial palsy that a technique was announced. At that time the repair of this condition was placed on a new basis. We are all familiar with this research as it was reported at the College of Physicians in Philadelphia by Duel[5] in 1933.

The technique developed by them is rational and affords the best method of re-innervating the face. Dr. Duel[4] says that caution is important in acquiring the technique. To quote, "I believe that those who attempt the work should so prepare themselves, by experience on the cadaver, that they would have a preconceived plan in their mind as to what is to be done in any particular case. Changes in technique which may prove any method may be conceived by experience. In my opinion such changes should be practiced on the cadaver 'propter hoc' and not 'post hoc'."

He further states, "A clinic should be established for these cases." This we firmly agree with, and it should be a clinic in which

such cases should be operated by one man and an assistant who devote their efforts in this direction. In Indiana University School of Medicine we have such a clinic and it is under the direction of Dr. B. E. Ellis.

The technique of this operation is well described by Tickle,[11] who was Dr. Duel's assistant. A short summary of the operative technique, according to Tickle, is as follows: Selection of cases are limited to those which involve the geniculo-foraminal subdivision of the nerve; first those which are due to accidental injury in operations on the mastoid; and second, those in which toxic infection or refrigeration of the facial nerve have produced a facial palsy. We have decided these cases should preferably be explored at once. This has the advantage that the segment of the facial nerve distal to the injury retains response to faradic current from 24 to 72 hours. Any exploration subsequent to this short period can only be an anatomical dissection. A preparation of the femoral cutaneous nerve can be made at once; after trying several other nerves for making a graft in seventh nerve paralysis, this one proved most suitable.

If the paralysis of the nerve occurs in this subdivision from toxicity or from exposure a decompression of the nerve is attempted.

The technique of approach is the same in either instance; the approach is made to the terminal subdivision of the nerve as it emerges from the stylomastoid foramen; then the geniculo-foraminal subdivision is uncovered from the stylomastoid foramen up to and proximally beyond the injury. This, Tickle says, is the least hazardous procedure.

In making a graft of the seventh nerve, asepsis of the external auditory canal is very important. The oozing should be stopped at the time of placing the graft but after the distal and proximal ends have been placed, subsequent ooze is not detrimental and is beneficial, since it helps fix the graft in position in a fibrinous clot which subsequently organizes.

Dressings are changed daily in badly suppurating cases, and every two to four days in non-suppurating cases. The wound is cleansed by instilling normal salt solution with a medicine dropper and removing the salt solution in the same manner. The final plastic closure depends upon each individual case.

It is problematical when the nerves begin to act, as shown by faradic current, but should it be even a very slight positive response, it indicates that there is a connection from the central nucleus to

the muscle, and even after this, one has to wait even weeks or months before voluntary action takes place. Where there is no response to galvanic stimulation, the patients are not advised to have this operation.

Bunnell[2] says, in the conclusion of his description of the repair of the geniculo-foraminal and terminal subdivisions of the facial nerve: "In the treatment of facial palsy emotional expression should be restored by repairing the nerve itself intratemporally or extratemporally instead of resorting to anastomoses with other nerves. This restores the control of the face by the emotional centers of the brain. If repair of the nerve is impossible, reconstruction of the face by a plastic operation involving the muscle and fascia is indicated. Decompression or neurolysis of the facial nerve often restores function. Direct union of the severed nerve ends by means of rerouting is preferable to the use of a free nerve graft when possible, because with it a more perfect degree of regeneration can be expected. Free nerve grafts should be used if the gap is too great for rerouting and will give good results. The degree of regeneration to be obtained in nerve repair is in direct proportion to the accuracy of the union of the nerve end. This argues for accurate, aseptic surgical repair of the nerve in a clean field by suture as against operation in the presence of infection, pus, free blood, open drainage and merely laying the nerve ends together."

The writer would make this comment: the technique for this operation is difficult and lends itself to much fewer cases than nerve grafts.

CONCLUSION

May we point out, then, in closing, that the three subdivisions as well as the two main divisions of the facial nerve, are important not only in connection with the etiological factors, but also with the treatment of seventh nerve paralysis.

608 GUARANTY BLDG.

BIBLIOGRAPHY

1. Bell, Sir Charles: The Nervous System of the Human Body, Embracing Papers Delivered to the Royal Society on the Subject of Nerves, 138, 1833.

2. Bunnell, Sterling: Surgical Repair of the Facial Nerve. Arch. of Otolaryng., 25:235-259, 1937.

3. Coleman, Claude C.: Surgical Treatment of Facial Spasm. Ann. of Surg., 105:647-657, 1937.

4. Duel, Arthur B.: Surgical Repair of the Facial Nerve Paralysis: A Clinical Presentation. 45:3-6, 1935.

5. Duel, Arthur B.: Advanced Method in Surgical Treatment of Facial Paralysis. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 43:76-88 (March), 1934.

6. Hunt, J. Ramsay: Herpetic Inflammation of the Geniculate Ganglion: A New Syndrome and Its Complications. J. Nerv. and Mental Diseases, 34:73, 1907.

7. Hunt, J. Ramsay: Geniculate Neuralgia. Arch. of Neurology and Psychiatry, 37:253-285, 1937.

8. Jefferson, Geoffrey, and Small, Ashton: Progressive Facial Palsy Produced by Intratemporal Epidermoids. J. of Laryng. and Otol., 53:417-443.

9. Kerrison, Phillip D.: Aural Survey of the Otitic and Technical Advances in Recent Decades. Amer. J. of Surg., 42:103-111.

10. Phillips, Gilbert: The Treatment of Clonic Facial Spasm by Nerve Anastomosis. Med. J. of Australia, 1:624-626, 1938.

11. Tickle, Thomas B.: After Care of Surgical Repair of the Facial Nerve. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 45:7-27, 1935.

XV

FIBROMAS OF THE NASOPHARYNX: JUVENILE AND CELLULAR TYPES

MAX L. SOM, M.D.*

AND

A. HARRY NEFFSON, M.D.*

NEW YORK

The purpose of this paper is to present the distinguishing clinical and histologic features between the juvenile angiofibromas and the cellular fibromas of the nasopharynx and to describe our experience in the treatment of four cases—two of the juvenile and two of the cellular variety. We are not concerned here with the small fibrous growths or the secondary fibrotic changes in inflammatory polyps of the nasopharynx.

The juvenile fibroma is a rare tumor which occurs chiefly in males between the ages of 10 and 25 years. It is very firm, almost cartilaginous, and, therefore, it should be easy to distinguish from the ordinary polyp which is soft. According to Schmidtmann, its point of origin varies. It may arise from the periosteum of the bony wall of the nasopharynx, occasionally from the under surface of the basi-sphenoid or the basilar process of the occiput, and less frequently from the spheno-palatine fossa, the foramen lacerum, the sphenoid sinus, or from the upper two cervical vertebrae. It is frequently difficult to determine the exact point of origin because of ulceration and the broadness of its base. Its growth is unlimited and it can extend into all the surrounding structures, whether they be soft or bony. From the nasopharynx it can invade the retromaxillary space, the posterior wall of the antrum, and in the cheek grow between the buccal mucosa and masseter muscle and through the muscle into the temporal fossa. It can also break into the orbit through the inferior orbital fissure. Growing forward into the nares, it can invade the adjacent sinuses, the inner wall of the orbit, or the hard palate. The great danger is the invasion of the base of the skull either by erosion of the bone or extension through the

*From the Laryngological Service of Dr. Rudolph Kramer and the Pathological Laboratories of the Mount Sinai Hospital, New York.

natural openings such as the cribriform plate, the superior orbital fissure, the optic foramen, or the foramen lacerum. Besides the signs of nasal obstruction, there can occur broadening of the nose, swelling of the cheek and exophthalmos. This appearance has been referred to as "frog face." Metastases are not observed.

In our two cases of juvenile fibroma, microscopic examination shows a predominance of star-shaped cells with tapering processes which represent immature fibroblasts. There are numerous blood vessels consisting of an endothelial lining surrounded by collagen fibers or embedded in a myxomatous framework. Elastic fibers and smooth muscle are noticeably absent. This lack of muscle wall probably accounts for the profuse bleeding encountered after biopsy, due to the inability of the vessels to contract. The myxomatous tissue which in Case 2 forms an integral part of the tumor may represent a differentiation of the immature fibroblasts into a myxomatous stroma. However, it cannot be denied that the immature fibroblasts and the myxomatous tissue may have a common mesenchymal origin. The same explanation holds for the rare occurrence of cartilaginous or bony stroma in this type of tumor. According to Schmidtmann, fatty degeneration is to be observed in the older patients, while in the actively growing tumors in young patients this is absent. Its presence is indicative of the beginning of regression of the tumor.

The etiology of the tumor is unknown. It is commonly ascribed to an inequality in the development of the bones of the skull so that in certain places the periosteum is unable to produce typical bone and, with the increase in the growth stimulus during puberty, hypertrophy occurs in place of bone formation. Ewing quotes Bensch as stating that the difference in the development of the male and female face and skull at puberty explains the predominance of the tumor in males. A remarkable feature of this growth is the spontaneous regression which seems to occur chiefly toward the end of the period when the tumor may develop, namely, after the twentieth year. The explanation for this may lie either in the natural anatomical changes at the point of origin from which the nutrition of the tumor is obtained, namely, calcification with consequent cutting-off of the blood supply, or, what is more likely, the disappearance of some endocrine growth factor, which has its counterpart in the fate of uterine fibroids following the menopause.

The cellular fibromas can occur at any age and do not appear to be governed by the factors of growth and regression that obtain in the juvenile fibromas. On microscopic examination the fibro-

blasts are seen to be mature and arranged in whorls in certain areas. Mitotic figures are nowhere to be found. The tumor is very cellular with a distinct fibrotic capsule. Some of these tumors, as in our Case 4, after several years may present a marked increase in cellularity, an abundant vascular network and occasional mitotic figures with slight pleomorphism. However, a definite diagnosis of sarcomatous transformation cannot be made.

A review of the literature shows that the treatment of these tumors varies. Formidable operations for their removal in regions inaccessible by the intranasal route are still reported with resulting high mortality and long morbidity. The injection of various chemicals and the use of electrolysis for the reduction in size and vascularity, followed by the removal of the shrunken tumor by the galvanocaustic loop have also been described, with somewhat better results. Electrocautery, employing both the monopolar and bipolar electrodes, has also been used with varying results. A great step forward has been the treatment of these tumors by radiation. This form of therapy has been employed by Allan, Schempp, New and Figi, Raynal, and others with encouraging results.

REPORT OF CASES

CASE 1.—I. G., a 17-year-old boy, was admitted to the Mt. Sinai Hospital on December 7, 1936, with a history of left-sided nasal obstruction and discharge with occasional headaches during the past two years, swelling of the left cheek for four months, and diminished hearing in the left ear for three weeks. On admission there was proptosis of the left eye and a non-fluctuant swelling of the left cheek and parotid region. There was no redness or tenderness. Pressure over the swelling caused saliva to exude from the opening of Stensen's duct. The left eardrum was thickened and red and the hearing was somewhat diminished. The inferior and middle turbinates on the left side were swollen and bathed in pus. There was a large, firm, irregular tumor completely occluding the left choana and overlapping the posterior margin of the septum and part of the right choana. There was a swelling of the left side of the soft palate. Roentgenologic examination showed clouding of the left antrum and ethmoids. A biopsy of the nasal growth was followed by profuse bleeding which required firm packing for a period of three weeks. The biopsy report was juvenile angiofibroma of the nasopharynx (Figs. 1 and 2). Microscopic examination shows a loose connective tissue consisting of a collagenous network made up mainly of fine fibrils with occasional denser fibers. Throughout the connective tissue groundwork are numerous small and large dilated vascular channels with a single layer of endothelium. Around many of these vascular channels the connective tissue appears concentrated, giving the illusion of a vascular wall, but no muscle or elastic elements are to be found. Under higher magnification the connective tissue cells appear large and immature. A noteworthy characteristic, in fact, one that we deem practically pathognomonic of this type of fibroma, is that they are usually stellate or angular. The cytoplasm extends into delicate processes merging with the surrounding collagen. The nuclei

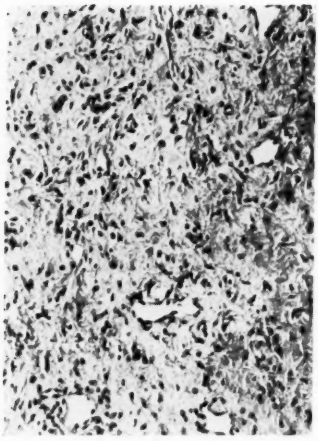


Fig. 1.

Figs. 1 and 2. Photomicrographs with low and high magnification of juvenile angiofibroma of the nasopharynx in Case 1. Note the large stellate and angular, immature connective tissue cells with the cytoplasm extending into delicate processes which merge with the surrounding collagen.

are large and either vesicular or hyperchromatic. Many of the cells are multinucleated. Another striking feature is the variability in the size and shape of the cells and the absence of mitosis.

The patient was treated by external radiation applied to the antrum and zygoma on both sides, giving a total of 2000 radon units over a period of one month. One week after the completion of the roentgentherapy, radium needles were implanted, giving him a total of 400 mg. hours of radium. This was repeated one month later when he received 720 mg. hours of radiation. The masses in the nose regressed but did not disappear. Two months after the last treatment radium was again implanted and he received 360 mg. hours of radiation. The proptosis of the eye, the swelling of the cheek, parotid region and soft palate, and the growth in the nose and nasopharynx gradually disappeared. Nine months after the last radium treatment only a small mass could be seen below and behind the left eustachian tube orifice; otherwise there was no evidence of the disease. He was symptom free.

Case 2. P. K., a 17-year-old boy, was admitted to the Mt. Sinai Hospital on December 23, 1931, with a history of chronic rhinorrhea and epiphora of two years' duration. One year before admission there was complete occlusion of the right nostril, for which he was treated with diphtheria antitoxin with no relief. Examination at that time revealed a growth in the nose with profuse, odorous discharge. A biopsy was taken and revealed a juvenile fibroma (Fig. 3). Micro-

scopic examination shows a myxomatous, relatively acellular groundwork containing numerous dilated, vascular channels. A single layer of endothelium constitutes the wall of these vascular channels. Around them the myxomatous tissue appears concentrated. Even under higher magnification the cellular elements in this myxomatous groundwork are sparse. The cells are irregular, angulated, with hyperchromatic nuclei and protoplasmic processes which extend into and merge with the collagenous groundwork.

According to his history, he then received an implantation of 12 radon seeds. However, since then he had several attacks of epistaxis and the foul discharge persisted. During the two months previous to admission, shaking chills had occurred every other day or so. Blood cultures were negative. Since the onset of his illness he lost 16 pounds in weight. On examination in the hospital there was evidence of a partial ethmoidectomy on the right side. There was a firm, reddish mass causing partial stenosis of the right choana. Roentgenography showed masses in the floor of the palate, on the right side, in the right antrum, and on the right side of the nasopharynx. Blood examination was negative. A Caldwell-Luc operation was performed and the posterior wall of the antrum was found to bulge forward due to erosion by a mass which on biopsy was found to be a juvenile angiofibroma. Radium was then implanted into the tumor, using five 5-mgm. seeds of radium element with a 0.5 mm. platinum filter. The seeds were allowed to remain for 24 hours, giving a total dosage of 66 mgm. hours. The symptoms then subsided.

Two months later there was a recurrence of shaking chills and fever, with swelling of the right cheek and pain in the right eye. Irrigation of the right antrum yielded sanguinous mucopurulent discharge. The fever slowly subsided. A month later he again developed febrile episodes with persistent swelling of the cheek and neck. An intranasal operation was performed and a tumor was found in the posterior ethmoidal region, apparently arising in an extension of the ethmoidal labyrinth into the posterior antral region. The mass was attached by a small pedicle to the right sphenopalatine ganglion region and extended medially so as to push the septum to the opposite side. The posterior third of the septum was removed and the mass severed at its attachment to the lateral nasal wall. The growth was 3 x 2½ cms. Following this procedure the symptoms subsided. Upon follow-up during the next two years there was no recurrence of the growth and he was symptom free.

CASE 3.—R. E., a 30-year-old woman, was admitted to the Mt. Sinai Hospital on June 18, 1933, complaining of pain behind the left eye and in the occiput for the past year, associated with nasal obstruction. On examination there was found a circumscribed, hard mass arising in the posterior wall of the nasopharynx, toward the left side, pushing the eustachian tube orifice laterally and extending downward so that it could be seen on the posterior pharyngeal wall for an inch below the uvula. The mucosa was movable over it. There was no infiltration of the surrounding structures and no glandular adenopathy. Through an incision of the pharyngeal mucosa the tumor was exposed and enucleated. It was found to measure 2 x 3 x 1½ cms. and was well encapsulated. There was another tumor in the region of the left lateral pharyngeal fold. On removal this was found to measure 2 x 1½ x 1 cms. The pathologist reported it to be a cellular fibroma (Fig. 4). Microscopic examination shows the tissue to consist of dark-staining spindle cells in whorl formation. In some areas these whorls have apparently become hyalinized. Under higher magnification the nuclei are seen to be vesicular and elongated, and

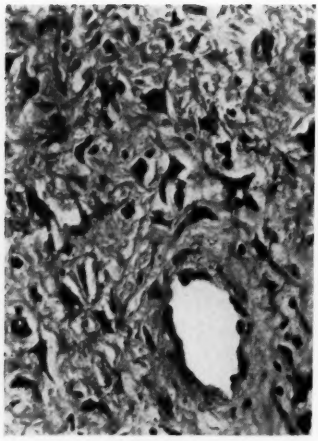


Fig. 2.

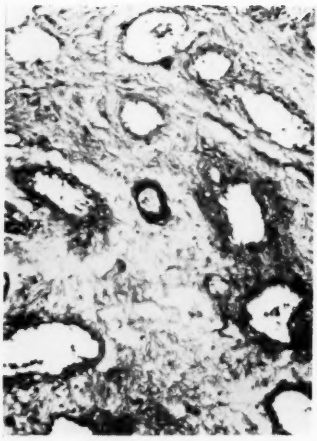


Fig. 3.

Fig. 3. Photomicrograph of juvenile angiofibroma in Case 2, showing a myxomatous, relatively acellular groundwork containing numerous dilated, vascular channels made up of a single layer of endothelium. Around many of these channels the connective tissue appears concentrated, giving the illusion of a vascular wall, but no muscle or elastic elements are to be found.

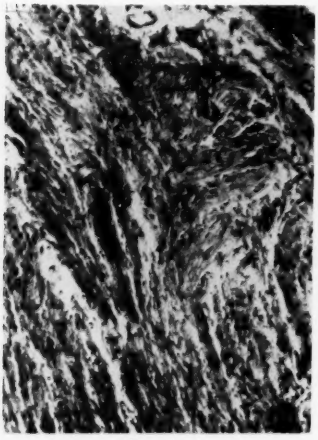


Fig. 4.

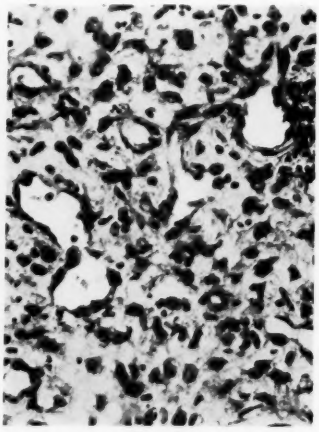


Fig. 5.

Fig. 4. Photomicrograph of cellular fibroma in Case 3, showing dark-staining spindle cells in whorl formation. In some areas these whorls have become hyalinized.

Fig. 5. Photomicrograph of cellular fibroma in Case 4, showing very cellular tissue made up of mature fibroblasts with large hyperchromatic nuclei and occasional nucleoli. The cells vary in size, are mostly rounded, occasionally bipolar. There are no mitotic figures. The star-shaped cells seen in Fig. 2 are not present here. There are numerous vascular channels consisting of a single layer of endothelium.

mitotic figures are absent. The patient was followed during a period of three and one-half years after the removal of the tumor. During this time she developed a mild sinusitis, but there was no evidence of recurrence of the growth.

CASE 4.—R. H., a 33-year-old man, was seen in the private practice of one of the authors. He stated that a fibroma had been removed from his nose five years previously because it had caused complete nasal obstruction and recurrent epistaxis. At the time of the initial operation he had bled profusely, requiring five transfusions and a stay of six weeks in the hospital. During this visit, September 9, 1937, he complained of nasal obstruction, purulent discharge and recurrent bleeding. On examination a large, firm mass was seen occupying the nasopharynx and presenting a rounded surface in the right posterior choana. It also extended around the posterior margin of the septum into the left choana. A biopsy was taken and was reported to be a cellular fibroma, quite vascular, with slight pleomorphism and occasional mitotic figures (Fig. 4). Microscopic examination shows a cellular tissue containing numerous very small and large vascular channels consisting of a single layer of endothelium. No true vascular wall can be discerned. The cellular components of the tumor are mature fibroblasts with large nuclei. These cells are mostly rounded and occasionally bipolar, and vary in size. The nuclei are hyperchromatic, and occasionally contain nucleoli. No mitotic figures are seen. The star-shaped cells previously described are nowhere to be found. There is evidence of chronic inflammation. A definite diagnosis of sarcomatous transformation could not be made.

He was given radiotherapy as follows: In the right nostril two long needles containing 6.6 mgs. of radium element and two short needles of 3.3 mgs. of radium element were inserted. A filter of 1 mm. of platinum was used. In the left nostril one long needle containing 6.6 mgs. of radium element and two short needles of 3.3 mgs. of radium element were inserted. In the nasopharynx cork containing 16.5 mgs. of radium element, distance 0.5 cm., and a filter of 2 mm. of platinum were inserted. These were allowed to remain for 32 hours, making a total of 15,840 M.G.H. Six weeks after the last treatment the tumor had shrunk to one-third of its original size. On the right side it had disappeared almost entirely, but on the left side it was still present. No further bleeding had occurred. He was able to breathe easily through his nose. There was no regional lymph node involvement. X-ray examination of the chest was negative.

COMMENT

In the treatment of the ordinary cellular fibromas of the nasopharynx, excision, with the capsule intact, is sufficient, as in Case 3. In Case 4 excision was impractical because of the large size and extension of the tumor. Its abundant vascularity would have been a further impediment to complete removal. The response to radium implantation was gratifying. Should no further reduction in the size of the growth occur, an attempt at extirpation by electrocoagulation will be undertaken. Because of the sclerosing effect of the radium, there should be no undue danger of profuse hemorrhage.

From a review of the literature and from our limited experience with the juvenile type, or angiofibroma, we feel that the

method of choice in the treatment is a combination of external radiation and the implantation of radium seeds. This is particularly so in those cases in which the tumor invades regions so inaccessible surgically as the retromaxillary space and base of the skull. If after this therapy there still remains some tumor tissue, surgical removal can be attempted, since the danger of profuse bleeding will have been minimized by the radiotherapy.

SUMMARY

A differentiation between cellular and juvenile fibromas, as regards the clinical, histological and therapeutic features, has been presented and two cases of each type have been described.

121 East 60th Street, New York City.

152 East 94th Street, New York City.

XVI

A REPORT ON THE USE OF UREA IN SUPPURATIVE CONDITIONS ABOUT THE EAR

A. P. Seltzer, M.D.

Philadelphia

The lack of any single agent on which the physician can depend to clear up the suppurative discharge in chronic conditions in and about the ear has led to the employment of various substances from time to time.

In reviewing the anatomy of the tympanum which (Gray[1]) "is an irregular cavity, compressed laterally, and situated within the petrous portion of the temporal bone. It is placed above the jugular fossa; the carotid canal lying in front, the mastoid cells behind, the external auditory meatus externally, and the labyrinth internally. It is lined with mucous membrane, is filled with air, and communicates with the mastoid tube."

The tympanic branch of the internal maxillary supplies the membrana tympani, while the stylomastoid branch of the posterior auricular supplies the inner wall of the tympanum, the antrum, and the stapedius muscle. The middle meningeal sends a branch to the tensor tympani muscle, while the petrosal branch of the middle meningeal enters the tympanum. Small branches from the posterior division of the middle meningeal are distributed to the antrum and epitympanic recess. The two tympanic vessels come from the internal carotid artery. The veins of the tympanum end in a plexis, the middle meningeal vein, and the superior petrosal sinus.

"The lymph channels of the membrana tympani itself are arranged in three systems, one for each layer. These communicate freely with each other and with the lymphatic network of the external meatus" (Dench[2]).

It has been established by Kopetzky, Preysing and others, that under normal conditions of life this cavity is kept sterile by the activity of the ciliated epithelium of the eustachian tube. The etiologic factors which result in an inflammation of the tympanum or its adnexa are pathogenic micro-organisms.

When, however, bacteria are at work, there are especial and characteristic differences in the exudate. There may be liquefaction of the exudate with necrotic tissue, depending on the causative agent and the solvent action of the micro-organisms.

Because of the poor anatomical blood supply of the tympanum, the spaces in the tympanum, particularly the incus, are prone to necrosis. When this poorly covered space is deprived of its protection, the bone readily succumbs to destruction and thus necrosis of the ossicles produces prolonged suppurative discharge of the ear.

The poor viability of that portion of the drum subjected to the greatest tension within, renders the drum more liable to perforation, thus spontaneous rupture of the most weakened part occurs, and from thence on the pus escapes into the external auditory meatus.

When prolonged otorrhea exists, an extensive repair is necessary, but sometimes the destruction of tissue is so severe that complete reparation is impossible. Primarily, free drainage from the tympanum is necessary, plus the maintenance of absolute cleanliness and local medicinal measures.

In the chronic cases of many years standing, antisepsis and drainage are necessary with surgical intervention, such as the removal of polypoid tissue, ossicles, or any other cholesteatomatous accumulations.

If purulent discharge cannot be completely drained from the tympanum, it at least must be rendered odorless and innocuous, for the comfort of the patient.

The difficulty of finding assured success in any treatment is easily understood by those familiar with the intricate anatomy of the field concerned, and its relative inaccessibility. Probably no location offers so secure a lodging place for pathological organisms, or a more favorable site for their growth, as the cavity of the middle ear and the mastoid cells, encased as they are in bony walls.

In recent years, the use of urea in the conditions of suppurative discharge in and about the ear has received considerable publicity in the literature of the subject.

This idea concerning urea primarily sprang from the work of Robinson,[3] who determined that the beneficial treatment of suppurative bone conditions with live maggots, which originated with Baer during the world War, was due to the hydrolysis of their excretions, and the consequent production of urea (Redenz[1]).

In 1906, before urea was used clinically in suppurative conditions, Peju and Rajat[5] studied its effect on bacterial growth in cultures. They found that there was less growth in the tubes containing urea, and polymorphic modifications also occurred. These results, however, required an optimum concentration of urea, and there was a variation in their results with different species of organisms. Certain ones were more easily modified than others. Concentrated solutions of urea have been found also by Wilson[6] to have a definite bactericidal effect in vitro.

Biochemically, Mathews[7] says that urea penetrates both animal and vegetable cells. In strong solution, it has been found to have unusual powers of dissolving proteins. It even dissolves such coagulated proteins as fibrin. Its behavior is that of an alkaline solution, though it is actually neutral in reaction.

In this relation, Heim[8] explains that the hydration of a protein solution is greater in the presence of urea than in urea-free solutions. These factors may apply to its influence on exudative lesions.

The biochemical effect of urea has been studied further by Hetherington[9] in relation to tissue culture growth. Here, no difference was found between the growth of explants in controls and in the presence of urea. Abel[10] has reported the finding of capillary proliferation, and this has been considered a source of benefit from the use of urea on infected wounds, thus stimulating the growth of granulation tissue.

The protective action of urea has also been reported in its power to destroy the viruses of acute poliomyelitis and rabies. MacKay[11] found that the virus lost its power of immunization in the presence of urea. The protein was denatured in undergoing the dissolving process.

Encouraged by the favorable reports of a number of physicians (Foulger and Foshay,[12] Mertins,[13] Bogart[14] Lewy,[15] and others[16]) on the use of urea in suppurative discharge from the ear, the writer began its use in the Brumm's clinic at St. Luke's Hospital, Philadelphia, as described by Mertins, employing a saturated solution on over twenty patients. The results have been uniformly disappointing. This lack of success in the use of urea can be clearly illustrated by the following cases:

REPORT OF CASES

CASE 1.—Martin R., age 34 years. Following a mastoidectomy, February 11, 1938, there was a facial paralysis, and a foul discharge from the ears. Bone necrosis was present. On February 28, 1938, the use of a saturated solution of

urea was begun, with instillation into both ears, after thorough cleansing. The patient was also given a saturated solution of urea to use at home, and was directed to put into each ear five drops three times a day. The foulness of the odor was lessened, but the discharge still continues, and is profuse though the use of urea is continued.

CASE 2.—Joseph J., age 11 years. Discharge from right ear since May 11, 1933. Everything possible has been done to clear up the discharge, but without success. In September, 1936, the right side was operated upon. On December 14, 1937, the use of a saturated solution of urea was begun, as described in Mertins' article. It was also given to the patient to use at home in five-drop doses four times a day. On February 8, 1938, the left mostoid was x-rayed and an operation was performed on the fifteenth. Following this urea was used until June 14, 1938. It was discontinued because of lack of beneficial results. The odor, though slightly lessened, is still foul.

CASE 3.—Mary G., age 14 years. Bilateral mastoidectomy was done on December 8, 1938, after several years of aural discharge, with increasing deafness. The discharge continued even after the operation. On January 13, 1938, the use of urea was begun in our clinic at the dispensary. Treatment was discontinued November 15, 1938, because of continued lack of benefit. The discharge remained just as profuse, and the odor, though slightly less foul, still continued.

In addition to the cases illustrated above, the writer has used urea in the after-treatment of polyps and cholesteatomata, as well as in acute and subacute conditions. It has also been made use of in the writer's private practice in nasal conditions, but still without success. Its use on granulation tissue following mastoid wounds has been equally unsuccessful.

In evaluating the results of the use of urea, it is interesting to notice that few have reported unfailing success in its use. Mertins admits "with few exceptions"; Foulger and Foshay find "almost all" are benefited; Lewy found it "apparently beneficial," and says further that "a more comprehensive study of this substance (urea) seems to be indicated to observe its value as a therapeutic agent," together with case reports indicating no benefit that could be definitely ascribed to the use of urea.

That those papers which have been presented as preliminary communications have not been followed by later conclusive reports, as far as the writer has been able to find, may be assumed to indicate that the continued use of urea has not been sufficiently successful.

In the literature its place seems to have been usurped by reports on the use of cod liver oil and other vitamin sources in the treatment of suppurating wounds.

Of particular significance would seem to be the fact that the use of urea in suppurative ear conditions has not been included in the recent textbooks of this subject.

The writer's own experience, together with the other evidence, strongly suggests that the local use of urea in otology has been another one of those straws which are frequently grasped with the hope that at last an effective agent has been found for the successful treatment of chronic discharging ears.

SUMMARY

1. The bactericidal and biochemical properties of urea are presented.

2. The literature dealing with the use of urea in purulent discharge from the ears is briefly noted.

3. Cases are presented from the writer's practice indicating an entire lack of success in the use of urea as a mode of treatment for suppurative discharge in and about the ear.

4. A final evaluation suggests that urea in otology has no therapeutic value.

1332 North Franklin Street.

BIBLIOGRAPHY

1. Gray's Anatomy (DaCosta and Spitzka), pp. 1026-1137. Lea & Febiger, Philadelphia, 1936.

2. Dench, E. B.: Diseases of the Ear: Lymphatics, p. 31. D. Appleton & Co., New York, 1909.

3. Robinson, W.: Use of Urea to Stimulate Healing in Chronic Purulent Wounds. Am. J. of Surgery, 33:192 (Aug.), 1936.

4. Redenz, E.: Der Harnstoff als keimtötendes Deoderans und seine Bedeutung für die Wundheilung. Muench. Med. Wchnschr., 85:1115, 1938.

5. Peju, G., and Rajat, H.: Note sur le polymorphisme des bacteries dans l'urée. Compt. rend. Soc. de Biol., 61:477, 1906.

6. Wilson, W. J.: Pleomorphism, as Exhibited by Bacteria Grown on Media Containing Urea. J. of Path. and Bact., 11:394, 1907.

7. Mathews, A. P.: Principles of Biochemistry, p. 264. William Wood & Co., Baltimore, 1936.

8. Heim, F.: Der Einfluss der Harnstoffs auf die Hydratation von Eiweiss. Biochem. Ztschr., 291:88, 1937.

9. Hetherington, D. C., and Shipp, M. E.: Effect of Urea Upon Growth of Fibroblasts from Cardiac Explants in Tissue Culture. Proc. Soc. Exper. Biol. & Med., 37:238, 1937.

10. Abel, R. G.: The Activities of Living Blood Capillaries in Relation to the Absorption of Urea. Anat. Rec., 67:1 (Suppl.), 1937.

11. MacKay, E. M., and Schroeder, C. R.: Virucidal Activity of Aqueous Urea Solutions. Proc. Soc. Exper. Biol. & Med., 35:74, 1936.

12. Foulger, J. H., and Foshay, L.: Antiseptic and Bacterial Action of Urea. J. Lab. and Clin. Med., 20:1113 (Aug.), 1935.

13. Mertins, P. S.: Use of Urea in Treatment of Chronic Otitis Media. Arch. Otolaryng., 26:509, 1937.

14. Bogart, L. M.: Urea: Its Use in Infections. J. Mich. Med. Soc., 36:285-287, 1937.

15. Lewy, R. B.: Use of Urea in Diseases of Ear, Nose and Throat: Preliminary Report. Arch. Otolaryng., 26:195 (Aug.), 1937.

16. Muldavin, L. F., and Holtzmann, J. M.: Treatment of Infected Wounds with Urea. Lancet, 1:540, 1938.

Baker, G. E.: Use of Urea in Treatment of Wound Infections. Rocky Mountain M. J., 35:310 (April), 1938.

Hoeder, H. G., and MacKay, E. M.: Use of Urea in Treatment of Infected Wounds. J. A. M. A., 108:1167-1169 (April 3), 1937.

XVII

CARCINOMA OF THE EXTERNAL AUDITORY CANAL, MIDDLE EAR AND MASTOID*

ROBERT J. BOWMAN, M.D.

BEVERLY HILLS, CALIFORNIA

Carcinoma of the external auditory canal, middle ear, and mastoid, is such a rare condition that even large clinics report very few cases, and it is possible for a busy otologist to see not more than one or two cases in a lifetime of practice. It is necessary, therefore, to study the literature on this subject in order to obtain a comprehensive picture which will enable the medical profession in general to arrive at a much earlier diagnosis of this grave condition. For here, as in carcinoma elsewhere in the body, early diagnosis with adequate treatment is most necessary if good results are to be expected.

On reviewing the literature one is struck by the oft-repeated statement that pain appears early in these cases and indeed is often the first symptom leading to a diagnosis of malignancy. One also gathers that malignancies of the middle ear and mastoid almost invariably terminate fatally despite extensive radical surgery and irradiation, while most of the cases which are reported cured will, on closer examination, be found to have been malignancies of the external auditory canal.

Malignancies of the external auditory canal are usually diagnosed much earlier than those occurring in the middle ear or mastoid, because the lesion is in a position where it may be readily seen and is ordinarily not masked by discharge from a chronic otitis media. When first seen by the physician, the patient usually complains of severe pain in his ear, with or without discharge, and possibly some hearing loss. Upon examination a tumor mass is found more or less occluding the lumen of the external auditory canal, and on manipulation is found to be friable and bleeds easily. It is difficult to believe that a carcinomatous mass of such dimensions could

*From the Department of Otolaryngology, The University of Southern California School of Medicine, Los Angeles, California.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, May 9, 1939.

be in a very early stage. Careful examination into the patient's history of his ear condition will almost always reveal that minor symptoms have been present for some time, often a year or longer, but did not annoy him enough to make him consult a physician until driven to it by pain.

The symptom of pain has been mentioned as an early finding by almost all writers who have mentioned it at all, and those who have tried to account for it have done so by assuming that it is caused by secondary infection when the tumor mass begins to break down. This belief is strengthened by the fact that a thin, foul secretion is usually present by the time pain is complained of and various organisms may be cultured from it.

On the contrary, it would seem that carcinoma of the external auditory canal is not in an early stage when the physician is first consulted, but that he may be led to think so by the briefness of the patient's history, which usually dates from the onset of pain. If this is true the pain complained of is not an early symptom and is therefore probably due to the same causes responsible for pain in carcinoma anywhere else in the body. The fact that growth of carcinoma in the middle ear, masked by the presence of chronic infectious discharge, usually progresses to the inoperable stage before pain becomes a marked symptom, strengthens this belief. However, the anatomy of the external auditory canal, together with the characteristics of the types of carcinoma usually encountered there, tend to limit the growth for a long period of time, so that metastases are almost never encountered. These factors, coupled with the easy accessibility of the lesion, are strongly in favor of a successful outcome following radical surgery, irradiation, or both.

The second, and by far the larger group, comprises those of the middle ear or mastoid region. In these the lesion has almost always progressed to great lengths before the diagnosis is made. Here the very commonness of chronic otitis media, and the great rarity of carcinoma combine to lead the physician astray. The great majority of writers agree that malignancy should be suspected in all cases that do not readily respond to routine treatment, including those in which polyps or easily bleeding granulations are found. This is a valuable recommendation, but when these features are present and through them the diagnosis is made, the carcinoma has almost always developed to a late stage. When the carcinoma originates in the mastoid itself, a correct diagnosis would be practically impossible until a very late stage has been reached.

The following case reports are submitted as illustrations.

REPORT OF CASES

CASE 1.—Mr. F. G., white, aged 69, entered the clinic on March 10, 1937, with the complaint of an intermittent discharge from his left ear for one year. He had been deaf in the left ear for the past few weeks. During this time he had severe neuralgic pains in front of the left ear, radiating to the temporal region.

On examination the right ear appeared normal. The left external auditory canal was filled with detritus and a mass of granulation tissue was seen on the posterior-superior canal wall in the inner third which greatly narrowed the lumen. A thin, very foul, serosanguineous discharge was present. There was no mastoid tenderness. Moderate adenitis of both anterior and posterior lymph chains was present on the left.

An x-ray of the mastoids showed no abnormalities. Hearing tests showed decreased air conduction and normal bone conduction in the left ear. A biopsy was taken from the granulating mass which showed a highly malignant epidermoid carcinoma, and at this time radiation was advised. The patient received 200 r. daily from March 25, 1937, to April 13, 1937, and then 150 r. daily until April 20, 1937, making a total of 3650 r. By May 6, 1937, the growth had entirely disappeared. On September 8, 1937, hearing test was normal in both ears.

When last seen in September of 1938, the left ear appeared normal. Whether this man is cured or not remains to be seen. He is intelligent and realizes that a recurrence may be grave, and therefore presents himself periodically for observation. If there should be recurrence, further radiation or radical surgery can be done early.

On entering the clinic, this patient's chief complaint was pain of five weeks' duration. The clinical examination showed a large mass practically occluding the lumen of the external auditory canal, and biopsies revealed a highly malignant carcinoma. Painstaking investigation into the history of the case showed that symptoms such as intermittent discharge, itching and "popping" of the ear, had been present for more than a year.

CASE 2.—Mrs. C. B., white, aged 59, entered the clinic on January 14, 1938, with the complaint of severe pain in the left ear for several months, together with a constant discharge. She had facial paralysis of seven weeks' duration. This patient had had a chronic left otitis media for forty years with many acute flare-ups. In November of 1935, her ear "popped" a great deal, and after January of 1936, there was occasional bleeding and "snapping" but no pain. In the summer of 1937, she developed mastoid tenderness followed by erysipelas of this region with uneventful recovery. During the three months before entry to the hospital she had severe pain around and in the ear and a constant profuse discharge. Seven weeks before admission she developed a sudden facial paralysis of the affected side.

On examination the right ear appeared normal. The left external auditory canal was filled with foul purulent discharge and was so occluded by edema that examination of the middle ear was impossible at the time. The mastoid tip was extremely tender and periosteal thickening was present. The hearing was markedly diminished. Cervical adenitis of the left anterior and posterior chains were present and a node one cm. in diameter was found above the left clavicle. An x-ray of the mastoids showed markedly diminished air contrast with hazy, partially obscured trabeculae, consistent with an exudative mastoiditis on the left. The hearing test showed a 40 per cent loss of air conduction on the left. The vestibular test was normal.

The swollen canal wall was treated with aluminum acetate wicks, and by January 26th the swelling was considerably lessened, so that granulations could be seen filling the middle ear and extending on to the posterior-superior canal wall. These bled easily on manipulation and a biopsy was taken which showed masses of squamous epithelium with cornified pearls and collections of chronic inflammatory cells. The epithelium appeared sufficiently hyperplastic to warrant a deeper biopsy, which was taken. The report on February 1st showed squamous cell carcinoma and radiation was advised to be followed by radical surgery. The patient received 3000 r. from February 14th to March 18th. On February 23rd a subperiosteal abscess over the mastoid tip was incised and drained.

The patient left the hospital on March 4th at the request of herself and relatives "to be built up to stand surgery." Operation was refused at that time but radiation was continued in the out-patient department. By May 3rd the ear seemed to have improved, the granulations were much less, but pain continued. At this time the supraclavicular node, mentioned before, had become quite large and painful. X-ray therapy was given to the extent of a total of 1800 r. from May 3rd to May 18th.

On May 26th the patient presented herself for admission for surgery, when it was discovered that she had erysipelas of the affected ear. She was treated in the contagious hospital and was returned to the ear, nose and throat department on the 30th.

On June 3, 1938, the approach for a left radical mastoidectomy was made. A large carcinomatous mass was found with destruction of the anterior, superior and posterior canal walls together with the attic wall. No structures of the middle ear could be identified. Most of the membranous canal wall was gone. The mastoid process was sclerotic. The cancerous tissue was removed as far as practicable and the wound was packed open so that radium might be used.

The patient received 533⅓ mg. hours of radium on June 16, 1938, and 543⅔ mg. hours on June 30, 1938, for a total of 1077 mg. hours.

On July 19th she left the hospital at her own request. She was seen four times in the clinic up to August 3, 1938, when she died at home. An autopsy was refused.

CASE 3.—Mrs. A. L. Y., white, aged 36, entered the clinic August 18, 1932, with the complaint of discharge from both ears for 33 years following an attack of measles. Impaired hearing was present in both ears. Bilateral simple mastoidectomy had been performed ten years previously. Both ears continued to discharge off and on since the operation.

On examination the right ear showed a postauricular scar. Crusts and mucoid discharge could be seen in the canal which, when cleaned away, showed a central perforation of the tympanic membrane. The left ear also showed a postauricular scar, and the tympanic membrane presented a marginal perforation when the mucopurulent discharge had been removed.

Both ear canals were cleansed twice weekly with peroxide and alcohol was instilled, followed by iodine powder insufflations. The right ear responded nicely to treatment and became dry in two weeks' time. The left ear continued to discharge and two months after treatment had been instituted showed a peculiar pyramidal mass of polypoid mucous membrane extending onto the lower posterior canal wall. A serosanguineous discharge was present. Granulations appearing in the middle ear were touched from time to time with 20 per cent silver nitrate.

A radical mastoidectomy was performed in February of 1934, and again in November, 1934, due to the continued accumulation of granulation tissue which was very friable and bled easily. This tissue was sent to the laboratory but the diagnosis was not made until January of 1935, after another biopsy had been taken, which showed squamous cell carcinoma.

The patient appeared to improve with deep x-ray therapy until six months later, when the tumorous mass had again enlarged so as to become troublesome. Three courses of x-ray had been used and further treatment was considered inadvisable. Death ensued and an autopsy was refused.

The advisability of early and, if necessary, frequent biopsies whenever the response to the usual treatment for a chronic ear with persistent granulations is at all extraordinary is clearly shown in this case.

It is obviously impractical to recommend that all chronic discharging ears should have radical mastoidectomies performed on them in the hopes that an occasional carcinoma might be averted. The only alternative is to keep the possibility of carcinoma, no matter how rare, in mind whenever treating a chronic ear. Bleeding is always a symptom which should make one suspect carcinoma.

Primary carcinoma of the middle ear or mastoid region, unaccompanied by previous chronic infection, has only been reported a few times in the literature. It occurs much more commonly in already chronically infected middle ears. As has been stated above, the infectious process often effectively masks the presence of the growth of carcinoma until a late stage has been reached.

Probably the condition which is most likely to be confused with carcinoma is tuberculosis of the middle ear. In this condition persistent, easily bleeding granulations may occur which do not respond to routine treatment. Clinically the appearance of the two lesions may be identical and therefore the laboratory must be relied upon for a correct diagnosis. In the final analysis the laboratory

must always be relied upon to make the diagnosis, as the physician can only suspect the presence of carcinoma by clinical observations and must confirm his suspicions by biopsy.

An interesting question is brought forth by the constant and continued use of concentrated silver nitrate on the persistent granulations following radical surgery in Case 3. Could this chemical trauma have been the etiologic factor responsible for the origin of the carcinoma?

SUMMARY AND CONCLUSIONS

1. Pain is not an early symptom of carcinoma of the external auditory canal, middle ear and mastoid region. The size of the tumor mass when pain appears shows that the carcinoma has been present for some time. Careful questioning of the patient will usually reveal minor symptoms which have been present for a considerable length of time before pain became a factor.

2. Tuberculous otitis media may simulate the course of a carcinoma so closely that only the laboratory may be relied upon for a correct diagnosis.

3. In all cases of chronic otitis media a possibility of the presence of carcinoma should be considered when the response to the ordinary course of treatment is at all unusual. Biopsies should be taken early and, if necessary, frequently in all cases where suspicious granulations exist.

4. The question is brought forth whether the use of concentrated silver nitrate or other caustics on granulation tissue of the middle ear and mastoid areas following radical surgery might be an etiologic factor in the origin of carcinoma.

415 North Camden Drive.

BIBLIOGRAPHY

1. Barnes, E. B.: Carcinoma of the Ear. J. Laryng. and Otol., 45:632-636, 1930.

2. Burton, F. A.: Epithelioma of the Middle Ear and Mastoid. Laryngoscope, 27:755-762, 1937.

3. Davis, H. J.: Epithelioma of the Middle Ear in a Young Man, Age 35. Royal Soc. of Med. Proceedings, 2:89, 1908-1909.

4. Fraser, J. S.: Malignant Disease of External Acoustic Meatus and Middle Ear. J. Laryng. and Otol., 45:636-643, 1930.

5. Goldbauch, J. S.: Squamous Epithelioma of the Middle Ear and Mastoid. Laryngoscope, 24: 128-131, 1914.

6. Guttman, J.: Report of a Case of Carcinoma of the Middle Ear. Laryngoscope, 30:727, 1920.

7. Law, E.: Deafness and Discomfort in the Right Ear as Early Symptom in a Case of Epithelioma Originating Near the Right Eustachian Tube. Royal Soc. of Med. Proceedings, 3:28-31, 1909-1910.

8. Milligan, W.: Carcinoma of Middle Ear: Facial Paralysis. Royal Soc. of Med. Proceedings, 5:26, 1911-1912.

9. Newhart, H.: Primary Carcinoma of the Middle Ear: Report of a Case. Laryngoscope, 27:453-555, 1917.

10. Risch, O. C., and Lisa, J. R.: Primary Carcinoma of External Auditory Canal and Meatus. Review of the Literature and report of a Case. Laryngoscope, 48:668-681, 1938.

11. Schall, L. A.: Neoplasms Involving the Middle Ear. Arch. Otolaryng., 22:548-553, 1935.

12. Whitehead, A. L.: A Case of Primary Epithelioma (?) of the Tympanum Following Chronic Suppurative Otitis Media. Royal Soc. of Med. Proceedings, 1:34-36, 1907-1908.

XVIII

A NEW LABYRINTHINE REACTION: "THE WALTZING TEST"*

CAESAR HIRSCH, M.D.

NEW YORK

When examining the labyrinthine function, i. e., the vestibular apparatus, with exclusion of the cochlea, we are accustomed to look first for the presence of nystagmus. Secondly, we seek to ascertain the presence of spontaneous past-pointing, or spontaneous deviation of the arms, on being stretched straight ahead in the horizontal plane. We further attempt to elicit Romberg's symptom, asking the patient to close his eyes and to bring toes and heels closely together. Animal experimentation, especially on the rabbit or, still better, on the guinea pig, whose body has a barrel-formed shape, shows that, on elimination of the labyrinth, this animal executes rolling or waltzing movements around its longitudinal axis at the climax of the reaction. However, we have never observed a similar phenomenon in human beings.

The following is a normal reaction to a simple test of equilibrium in the human being. The subject is asked to close his eyes, to stretch his arms straight before him and then to flex and raise first one knee and then the other, and so forth, thereby passing from a stable to a more labile position (Fig. 1). The normal individual is capable of performing the test not only without loss of equilibrium but also without any subjective sensation of vertigo. This manever is based upon the same principle as Jendrassik's maneuver for emphasizing the patellar reflex, i. e., exclusion of the inhibitory effect of consciousness upon an involuntary reaction. Were any of us to conduct the same experiment in a starting, moving, or stopping elevator, a certain disturbance of equilibrium would be felt. The subject tested above would of course not be told what his reaction may or may not be. Not only must his eyes be closed during the entire experiment, but he should not even be spoken to, in order to avoid the possibility of acoustic orientation of his position.

---

*Paper read before the New York Neurological Society in the New York Academy of Medicine on October 3, 1939.

Fig. 1.

If, however, the vestibular apparatus is stimulated in the usual way by the caloric, rotatoric, galvanic, or mechanical method, thereby producing a nystagmus, the subject will act differently when placed in the above described position and asked to lift first one leg and then the other. After a certain interval (latent period) the arms of the subject begin to deviate to one side in the direction of the slow component of the nystagmus, whereby the external arm descends somewhat from the horizontal plane. Soon the subject begins to spin around his vertical axis, following the direction of the deviation of his arms. This spinning shortly thereafter takes on the form of waltzing movements, since the subject's body follows a sensation of traction or pull in the direction of the stimulated labyrinth and the slow component of the nystagmus respectively (Fig. 2). Depending upon the sensitivity of the subject (or, as it has also been called, the readiness for nystagmus) and upon the intensity of the irritation, a normal individual will spin around on his vertical axis for about 360 degrees and may sometimes return to his original position.

There are, however, a number of factors to be observed: First of all, among all the methods of stimulating the vestibular apparatus, the caloric test is the most convenient. Furthermore, the stim-

Fig. 2.

ulation of the labyrinth must be performed in such a manner that the reaction does not become too severe. If one stimulates the labyrinth of an individual with such intensity that he loses his balance entirely and cannot remain upright, it is obvious that one cannot expect him to spin on his vertical axis.

It is for this reason that the so-called "weak stimulation method" must be used. If one considers the normal body temperature of an individual to be 37° C. (98.6° F.) then 5 cc. of water at a temperature of 27° C. (80.6° F.) or 47° C. (116.6° F.) are thus able to produce in most individuals a very pronounced nystagmus, past-pointing, tendency to fall and subjective vertigo. Even when using the so-called minimum caloric irritation, which means instillation of one cc. of water at a temperature of 36 or 38 degrees C., some individuals may show all of the above described labyrinthine reactions, in a very mild degree, however. Our investigations have shown that the waltzing reaction is best produced by using five cc. of water at a temperature of 15 degrees C. (59.0 degrees F.).

If one irrigates the external canal of the ear of an average human being with five cc. of water at a temperature of 15 degrees C. and brings the plane of the horizontal semicircular canal in the optimum position by raising the head 60 degrees, so that the ampulla of

Fig. 3.

the external semicircular canal stands above and the straight end below, an average normal individual will show the first signs of deviation of the arms after approximately ten to fifteen seconds. Twenty to thirty seconds elapse before the first signs of spinning in the direction of the irrigated ear (i. e., the slow component of the nystagmus) appear, provided that the nystagmus is observed. After several seconds the entire body of the individual begins to show a postural deviation in the direction of the stimulated ear, and a more pronounced waltzing movement will occur. This waltzing movement will continue as long as the stimulation of the labyrinth continues.

In some cases there may occur a complete waltzing revolution of 360 degrees, while in others it may cease at 270 or 180 degrees. Much depends upon the individual sensitivity of the subject and upon the time which elapses until the original temperature of the external auditory canal is restored, following the irrigation. (We know, for instance, that after irrigating the external auditory canal with five cc. of water at a temperature of 27 degrees C. the original temperature in this external auditory canal will be restored after approximately 160 seconds.)

If, on irrigating the right ear with cold water, one changes the position of the stimulated labyrinth in such a way that the stimu-

Fig. 4.

lated right labyrinth is turned 90 degrees, so that the subject's nose is directed to the left and his ear straight forward, the deviation of the body takes place in the direction of the stimulated labyrinth—namely, forward instead of sideward, as would happen if the patient's head were kept in the normal position (Fig. 3). In such a case the patient has the subjective sensation of being pulled forward.

If the stimulated right labyrinth is turned backward, so that the subject's nose is directed to the right, the deviation takes place backward, and the individual has the feeling of being pulled backward.

However, under both conditions, the waltzing always takes place in the same direction, namely, in the direction of the slow component of the nystagmus. The person is never aware of the fact that he spins around. He may, however, have a "swaying" or "swimming" sensation.

If on stimulating one labyrinth with cold water the head of the subject is tilted 90 degrees to the left shoulder or to the right shoulder, so that the plane of the horizontal semicircular canal is placed in a "pessimum" position, no waltzing will be observed (Fig. 4). Thus the waltzing test appears to be a special means of testing the function

of the horizontal semicircular canal alone. The pathway over which the waltzing reaction travels is in all probability the vestibulo-spinal tract. Animal experiments will demonstrate whether there are other tracts involved such as, for instance, the vestibulo-rubral tracts and the rubro-spinal tracts respectively.

As far as the pathology is concerned, I should like to state briefly that, of course, in an individual with a dead labyrinth the waltzing reaction cannot be elicited. On the other hand, the waltzing reaction may easily be observed as a spontaneous reaction in patients suffering from a disturbance of equilibrium, whether it be due to decompensation following irritation or deficiency. If, for instance, on stimulation of the labyrinth only falling and past-pointing is elicited but no nystagmus, there exists the probability that the lesion lies in the fasciculus longitudinalis posterior. If nystagmus, past-pointing, and falling but no waltzing is elicited, there exists the probability that the lesion lies in the vestibulo-spinal tract. If past-pointing alone is produced, the other reactions being normal, the lesion is probably located in the cerebellum or efferent cerebellar pathways. If an existing spontaneous deviation ceases following the stimulation of the vestibular apparatus, we may assume that cerebellar irritation is the cause of the past-pointing.

REPORT OF A CASE

CASE 1.—Mrs. S. J. C., a cartoonist, 36 years of age, was suffering from an acute otitis media on the left side, complicated by dizziness, a sick feeling in the stomach, general lassitude, and vomiting. No spontaneous nystagmus was present.

Waltzing test showed a spontaneous spinning of the body towards the left of 35 degrees.

Caloric test on the right ear with five cc. of water of 15 degrees C. showed, after a latent period of 34 seconds, a horizontal rotatory nystagmus of 112 seconds duration, with past-pointing +, tendency to fall +, subjective vertigo +. Waltzing test showed, after a latent period of 15 seconds, deviation of the arms towards the right, and after 30 seconds waltzing of the body approximately 270 degrees towards the right.

The caloric reaction on the left ear showed, after a latent period of 19 seconds, a very intensive and more frequent horizontal rotatory nystagmus towards the right, of 221 seconds duration. Past-pointing + +, tendency to fall + + +, subjective vertigo + + +. After a latent period of 10 seconds, the patient showed deviation of the arms, after 20 seconds patient started waltzing towards the left, four times 360 degrees, and vomiting.

After myringotomy and washing of the ear with a three per cent solution of hydrogen peroxide and application of heat, the purulent secretion from the left middle ear diminished, and the drum closed. The patient suffered from a

severe feeling of dizziness and vomiting which disappeared after the use of Vasano suppositories. Only on one day, and that was four days after the beginning of the symptoms, a slight spontaneous horizontal rotatory nystagmus towards the right was present, so that in this case the waltzing reaction was an earlier objective symptom of the disturbance of equilibrium than the disturbance of the oto-ophthalmic apparatus.

An audiogram after the closure of the eardrum showed normal hearing in the diseased ear up to 2048 cycles, with a diminution for higher pitches: 20 decibels for 2896 cycles, 40 decibels for 4096 cycles, 45 decibels for 5792 cycles, 75 decibels for 8192 cycles, and 95 decibels for 11584 cycles. In the course of about three weeks the subjective symptoms disappeared completely, while the patient still showed a spontaneous waltzing reaction towards the diseased (left) side of about 60 degrees.

SUMMARY AND CONCLUSIONS

The waltzing reaction is elicited by stimulation of the horizontal semicircular canals. It is best produced by a caloric stimulation of the labyrinth with five cc. of water at a temperature of 15 degrees C.

The waltzing reaction is probably due to stimulation of the homolateral vestibulo-spinal tracts.

With the help of the waltzing test it is possible to make a topical diagnosis of lesions of the posterior longitudinal bundle in the floor of the fourth ventricle. The waltzing reaction may in some cases be an earlier symptom than spontaneous nystagmus.

The waltzing test is superior to observation of the oto-ophthalmic apparatus, because it can be observed much more easily, the reaction can be photographed in each case, specially filmed, and thus kept on record much more easily than a nystagmus.

The waltzing test does not inconvenience the patient.

667 MADISON AVENUE.

XIX

AN ANATOMIC "CROWN"

AS AN AID FOR POSTGRADUATE STUDY OF THE ANATOMY OF THE HEAD

SOL. MALIS, M.D.

LOS ANGELES

In presenting this paper the writer has in mind the hundreds and probably thousands of otolaryngologists who for some reason have no opportunity to visit centers where postgraduate courses are offered, and who yet would like to keep fresh in their memory the anatomy of the head. To this end a method is suggested for securing a permanent specimen for such study and demonstration.

However small the town may be where such specialists are found, there are always mortuaries where autopsies are made. At the mortuary the physician may secure such a specimen, as authorized by state law, without any objection on the part of the undertaker. There is no damage done to the face during the removal of such a specimen, and the external carotid arteries are preserved for embalming purposes.

TECHNIQUE

The instruments necessary for such a dissection are usually found at every mortuary, with the exception, perhaps, of a nasal speculum, and a keyhole hacksaw, which is easily procured at any hardware store.

The scalp and epicranial aponeurosis are incised from ear to ear at a point one inch behind and one inch below the upper border of each auricle, along the bregma. The anterior half of the scalp is then pulled well down over the face to about one inch above the superior orbital margins; and the posterior half, well down over the nuchal region (external occipital protuberance). The temporal fascia and muscle are scraped downward. The calvarium (skull cap) is then removed with a saw in a circular manner, to the extent of the reflected scalp. In lifting the calvarium it may be found to be adherent to the underlying dura; this is to be gently separated. The dura is then incised in a crucial manner, making one incision immediately along the side of the superior sagittal sinus, and the

other similar to the primary incision of the scalp. The flaps thus formed are turned down. The falx cerebri is then detached from the frontal crest, and is withdrawn from the cerebral hemispheres. The brain is removed antero-posteriorly, raising the frontal lobes from the anterior cranial fossa, the cranial nerves identified and cut long to the skull with scissors, for the purpose of subsequent study. At the posterior fossa the tentorium is cut on each side along the antero-posterior borders of the petrous bone to dislodge the cerebellum.

The Meninges and the Venous Sinuses: At this point one might review the membranes enveloping the brain. They are the dura mater, arachnoid mater, and the pia mater; their names indicate their qualities: the dura is tough, the arachnoid is like a spider's web, and the pia clings faithfully to the brain surface like a skin, following all its irregularities. Between the dura mater and the arachnoid mater is a potential space, the subdural space. Between the arachnoid mater and the pia mater there is an actual space, the subarachnoid space, filled with cerebrospinal fluid. The arachnoid mater is attached to the pia mater by loose scattered threads. As the arachnoid does not dip into the sulci of the brain, but bridges them, innumerable small and several large cisterns filled with cerebrospinal fluid are seen in the carefully removed brain. The cerebral arteries travel in the subarachnoid space.

The cisterns, particularly those that are situated at the base of the brain, are of immense importance from a clinical viewpoint. In early meningitis of otitic origin, following mastoidectomy, many a case has been saved by incising the dura and arachnoid of the adjoining cistern to allow for the escape of the involved cerebrospinal fluid.

A point of interest is to note the reduplication of the four inwardly projecting folds of the dura mater; the sickle-shaped occupants of the median sagittal plane, the falx cerebri, and the falx cerebelli; the roof of the cerebellum, the tentorium, and the roof of the hypophysis cerebri, the diaphragma sellæ. These partially subdivide the cranial cavity into compartments, and being taut they prevent shifting of the cranial cargo, which is the brain.

Another point of interest to the otologist is the venous sinuses of the dura mater, particularly the lateral sinuses which are severed during the removal of the "crown." This is the occasion to examine their formation, connections and continuation. Whether the right lateral sinus is the larger and whether it is a continuation of

the superior sagittal sinus, or branching out from the confluent sinus at the torculi. Each lateral sinus should be incised in its entire course, its width and lumen examined, as well as the absence of valves noted. Note also that all sinuses drain ultimately into the internal jugular veins.

Removal of "Crown": To facilitate sawing, the peripheral dura is now separated from the skull and excised, leaving the central attachment for the preservation of the cranial nerves and for further anatomic dissection.

An outline of the "crown" to be removed is first traced with an indelible pencil on the base of the skull. Beginning anteriorly with the foramen cæcum, a curved line is drawn over the roof of the orbit to a point of junction of the lesser and greater wings of the sphenoid bone, and continued along the squamous portion of the temporal bone and extended backward on the petrous portion one-half inch posterior to the arcuate eminence, so as to include the inner and middle ear with its drum, and then posteriorly, to the jugular, and into the foramen magnum. A similar outline is made on the other side. The purpose of this tracing is to guide us in the sawing which is to follow.

If an Albee circular saw is on hand, superficial serrations may first be made and followed up by the keyhole hacksaw. The tip of the hacksaw is now entered through the thin roof of the orbit, and the removal of the "crown" is begun. With the handle of the saw held obliquely outward and laterally to the superior orbital fissure, the greater wing of the sphenoid bone is bisected, cut through the floor and outer wall of the orbit, when the tip of the saw will be felt to enter the maxillary sinus, and extended backward through the squamosa and the petrous bone. Some resistance may be met here both on account of the hardness of the bony structure and the clogging up of the saw with bony filings. To obviate this the saw is removed from time to time and the filings shaken off in water. As the petrous bone is sawed through, the cut is continued along the occipital bone posterior to the jugular foramen, so as to include the latter as well as the canalis hypoglossi, into the middle of the foramen magnum. The other side is similarly cut through as far as the foramen magnum, which is later on dislocated and elevated from the atlas.

The nasal septum is now resected from the basis cranii internum. Beginning at a point of the first cut of the saw through the thin roof of the orbit, the sawing is continued across the fora-

men caecum, anterior to the crista galli of the ethmoid to meet the cut in the roof of the orbit on the other side. In cutting through the septum, the saw should be held parallel to the bridge and the tip of the nose, carrying the incision as far as the mucocutaneous junction within the vestibule, but not through the columella. At all times care should be exercised to avoid perforating the skin of either lower eyelid. Only the tip of the saw should be utilized here, and with every few motions of the saw, the skin of the face should be inspected, to prevent perforation. The saw will be felt to give after the septum is cut through. At this point we find that not only the septum is cut through, that both middle turbinates and ethmoidal labyrinths are severed anteriorly from the nasal processes of the superior maxillae, but also that the inner walls of both orbits are retained with their laminae papyracea. The nasal vestibule is now widened with a nasal speculum, and a small knife is inserted through the septum at a point of the last cut of the saw, and carried to the floor of the nose to the anterior nasal spine, as for submucoid resection. With the same knife, about one inch of the cartilaginous septum is resected from its floor to allow for the insertion of the tip of the saw to cut through the cartilaginous and osseous portions of the septum from the floor of the nose in its entirety, into the nasopharynx. The tip portion of the cutting saw is continued along the floor of the nose, and the lateral nasal wall below the inferior turbinate is thus severed, beginning posteriorly into the middle pterygoid lamina, through the pterygoid fossa, and through the lateral pterygoid lamina into the subtemporal fossa. At this point the saw will be felt to cut through the maxillary sinus at its floor. This is continued to meet the vertical cut posteriorly behind the orbit, and anteriorly in front of the inferior turbinate. The saw is now removed and passed under the detached septum along the floor of the nose, on the other side, under the inferior turbinate, and the lateral wall of that side of the nose similarly severed. In sawing through the lateral nasal wall, care should be taken not to nip the skin of the vestibule of the nose. To obviate this, the wider two-thirds of the saw is well covered with gum paper or adhesive. Before beginning with the delivery of the "crown" it is well that the incisions thus far carried out are made complete.

Delivery of the "Crown": The delivery of the "crown" is now undertaken. The atlanto-occipital joint is severed with knife and scissors, and a strong hook is inserted anteriorly between the foramen magnum and the atlas, and traction exerted on the hook, upward and forward. While this is being done the muscles of the

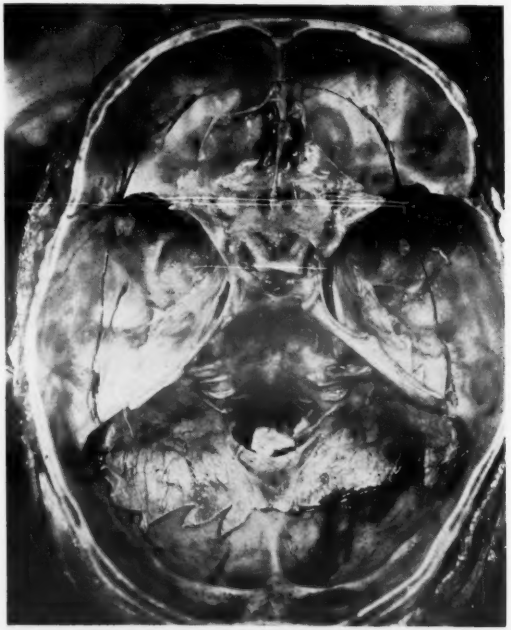


Fig. 1.

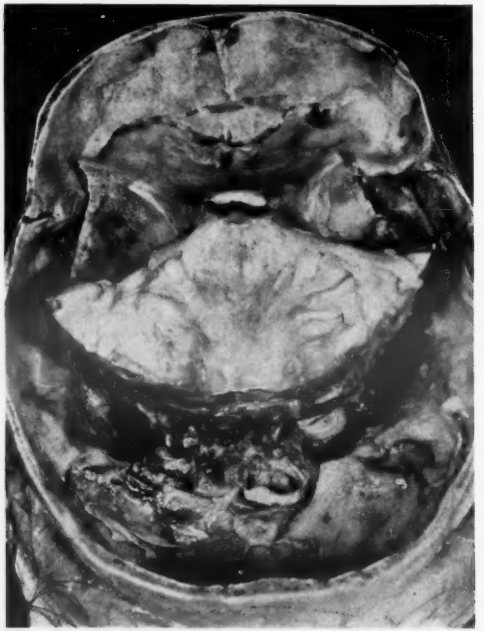


Fig. 2.

Fig. 1. Photograph of basis cranii internum, showing serrations into skull with central dura intact.
Fig. 2. Basis cranii internum, showing specimen to be removed.

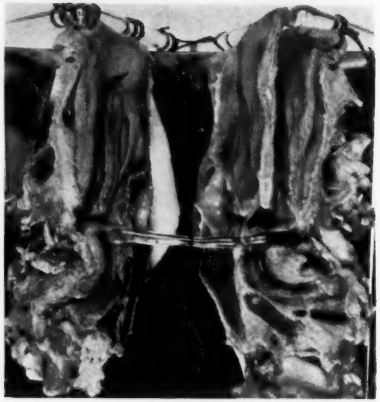


Fig. 3.

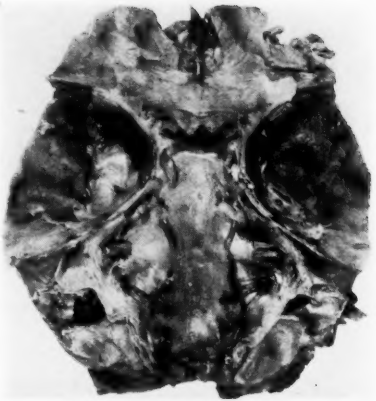


Fig. 4.

Fig. 3. Sagitally sectioned specimen suspended on glass rod with black silk.

Fig. 4. Removed specimen showing superior surface with peripheral dura removed and all cranial nerves intact.

base of the skull as well as the fascial attachments are severed with knife and scissors until the entire "crown" is loose, as shown in Fig. 2. The specimen thus obtained assumes the shape of a "crown," the upper portion of which is formed by the inner surface of the base of the skull, and the lower portion by the septum, turbinates and maxillary sinuses. The specimen is now gently washed in cold running water and placed in Kaiserling solution No. 1*, either in toto or sectioned sagitally. The sectioning is carried out immediately laterally to the frontal crest along the cribriform plate, so as to retain the septum intact on one side, while the other side exposes the lateral nasal wall with its turbinates, as in Fig. 3. The specimen may remain in solution for one to five days, or longer, when it is removed and placed again in running water for 24 hours. It is then immersed in 95 per cent alcohol for two to five hours to restore its color, which is affected in the fixing solution; and finally, for office use, the "crown" may be suspended on a glass rod by a strong thread within a museum glass jar, 6 x 6 x 4 inches, containing Kaiserling solution No. 3**. The advantages of using the Kaiserling (solution) method over the old formalin are the following: It does not dissolve the bone; it does not burn or harden the tissues; and it is not pungent to the eyes during later dissection and study of the specimen.

Having thus delivered the "crown," every vital structure of interest to the otolaryngologist, with the exception of the frontal sinuses, is preserved. Posteriorly we have a portion of the bony ear canal, the intact inner and middle ears with their drums, the mastoid and its antrum, the entire course of the facial nerve within the temporal bone, as well as the greater superficial petrosal nerve; all cranial nerves; above and in the center are the cavernous sinuses with their contents; in front we have the posterior halves of the orbital cavities, the septum, and all turbinates; and laterally, both antra and ethmoid labyrinths. Inferiorly and laterally, one will note the infratemporal fossa with the infraorbital fissure and the structures passing through the latter, and more medially, the contents in the pterygoid fossa, its laminae and their relationship to the posterior nares; whereas postero-inferiorly, on each side, one will find the eustachian tubes in toto, and in the middle and above, the sphenoid sinuses with their ostia.

---

*Kaiserling Solution No. 1:
Formaldehyde (commercial), 200 cc.
Water, 1000 cc.
Potassium nitrate, 15 grams.
Potassium acetate, 30 grams.

**Kaiserling Solution No. 3:
Potassium acetate, 100 grams.
Glycerin, 200 cc.
Water (sat. with thymol), 1000 cc.
Formalin, 1%.

In further dissection of the "crown" one will also find all ganglia of interest to the otolaryngologist, particularly the gasserian, the geniculate, the sphenopalatine and the otic, with the carotid sympathetic plexus, as well as the position of the drums, the interior of the middle ears, their dimensions and contents. On the inferior surface of the "crown" one will further note the cranial nerves and vessels as they emerge from their foramina, and their relationship to one another. Of particular interest is the terminal portion of the internal maxillary artery, the spheno-palatine, as it passes through the spheno-palatine foramen into the nasal cavity, giving off branches to the septum, lateral nasal wall, the sphenoid sinuses, and the antrum. Nowhere else could the position and course of this important vessel be studied and appreciated as in such a specimen.

SUMMARY

The advantages of possessing such a "crown," instead of studying anatomy on the cadaver, are several: (1) the structures are all intact and their relationship better understood; (2) the specimen is fresh, unlike the one on the cadaver, which is hard and friable; (3) and it is always on hand for review purposes. If skill is acquired in removing such "crowns," one may secure several specimens for the study of variations and anomalies, as well as specimens of the mucous membranes of the sinuses, both gross and microscopic.

The possession of such a specimen affords one an opportunity for research: the study of anomalies of the bones, and vessels, and dehiscenses, especially those of the middle ear, and those in the neighborhood of the sphenoid sinuses. For the rhinologist, the sagitally sectioned specimen unlocks many hidden vistas. Here he can see, as nowhere else, the os cribrosa, the olfactory prolongations, and the so-called danger zone for intranasal operations; the superior meatuses within the nose, the relations of the posterior ethmoidal cells to the sphenoid sinuses and to the optic foramina.

With the recent entity of involvement of the os petrosa in infections of the middle ear, not only can the otologist familiarize himself with the gross and microscopic cellular structure of this bone, but it is possible for him to study the different surgical approaches to the petrous pyramid, as well as understand the petro-sphenoidal articulation, the so-called Dorello's canal, and appreciate Gradenigo's syndrome.

It is a common fault of technique of the radical mastoidectomy not to destroy thoroughly the tensor tympani attachment and the processus cochleariformis. Only by a more thorough knowledge of these structures, and by having easy access to the middle ear and tympanic portion of the eustachian tube, will the otologist remember this anatomic region, and visualize the course of the internal carotid artery and its relationship to the eustachian tube, and will ever bear in mind how to avoid injuring this important vessel in the curettement of the tube.

CONCLUSIONS

One need not emphasize the importance of the knowledge of anatomy to the otolaryngologist. In his daily work he is confronted with problems affecting any and every vital part of the head; and by bearing in mind the development, and the pathological processes, whether they be inflammatory, developmental, vascular, or neoplastic, he is enabled to better understand and interpret the clinical picture.

1032 ROOSEVELT BUILDING.

This specimen is of advantage for the study of the minute anatomy of the membrana tympanic, its position, thickness, and actual attachment to the annulus tympanicus, particularly in the Lempert operation for otosclerosis.

I do not claim originality for the idea of securing such a specimen; but the title, the purpose, the modification of the technique, its description, and its practical application, as they appear in this paper, are entirely mine, arrived at over a period of years of research—since 1929, when I assisted Dr. Wells P. Eagleton in his first case of "Unlocking of the Petrous Pyramid" (Archives, March, 1931). T. Wingate Todd, Sewall and Hunnicutt may have suggested it, notwithstanding that, to my knowledge, the method has never been published.

XX

THE TYPE OF DEAFNESS WHICH RESPONDS TO FISTULIZATION OF THE OTIC CAPSULE*

NORTON CANFIELD, M.D.

NEW HAVEN, CONNECTICUT

In the socializing process of human relations, good hearing is secondary only to intelligence. At last this special function is beginning to receive its due consideration from both the laity and the medical profession. Complicated by the manifold factors involved in the restoration of hearing, investigation is now being directed toward specific problems which are gradually yielding to methods which have been developed as science has progressed. The recent encouraging interest in the subject of deafness demands that we clarify as soon as possible the muddled ideas which now exist in regard to the type of treatment to advise for our deafened patients. It is of little concern to those who come to us for help as to what part of the ear is affected, whether their disability is due to a so-called conductive or a perceptive lesion. These patients find themselves socially unadjusted because they do not hear well enough and they seek any advice which offers hope of re-establishing this important function.

I wish to present the clinical picture of patients who will respond to the fistulization operation. I will point out types which have not responded to this procedure. We do know that if the patients fall within a rigid group, a fistula does something to the hearing mechanism so that air-borne sounds are heard at lower thresholds. About this there can be no doubt. Such careful observers as Jenkins,[1] Barany[2] and Holmgren,[3] and any of you who have done or have seen the operation performed are convinced that, temporarily, hearing is improved after the fistula is made. These people hear sounds which they did not hear before. Associations in their daily life are changed. The sound movies, the radio and ordinary conversation are heard better in enough patients to remove any suspicion of a

*From the Department of Surgery, Division of Otolaryngology, Yale University School of Medicine, New Haven, Connecticut.

Read before the Middle Section of the American Laryngological, Rhinological and Otological Society, Incorporated, Kansas City, Missouri, January 19, 1940.

pure psychological phenomenon. The improvement of these patients is confirmed by their associates, and others seek the same type of help.

What findings, then, must these people present to fall within this group, and how far can we deviate from this selected group and still offer hope?

If we accept the present theory that a new opening into the otic capsule permits sound waves to produce an effect upon the cochlear fluid, thereby enhancing the stimulation of the organ of Corti, it is hard to imagine anything but a mechanical force which does it. We believe that a vibrating basilar membrane is essential to stimulate the hair cells. When these vibratory movements are diminished, as is the case when the footplate of the stapes is fixed to the oval window, the threshold for sound perception is changed so that more external power from the sound source is necessary to produce the same cochlear effect. The hearing defect can often be alleviated either by increasing the incoming sound above the patient's threshold as is the case when one speaks louder, or by lowering the threshold as is the case after the fistula is made. This presupposes that the rearranged inner ear with a new fistula does not improve either the microphonic effect of the cochlea itself, or the neural mechanism which transmits the impulse to the brain.

It is known that in normal ears during sound perception the round and oval windows are vibrating in opposite phase. That is, when the stapes footplate is pressed into the labyrinthine fluid by a sound wave impulse, the round window membrane bulges into the tympanic cavity. With the annular ligament fixed, more power is necessary to depress the footplate. Hence, less movement of the endolymph results and less movement of the basilar membrane and the round window occurs. If a third opening is made into a bony otic capsule whose oval window is fixed, there is now a new possibility for endolymph movement. It is supposed that the fistula provides the means whereby the vibratory movements of the endolymph are greater with the same volume of sound pressure. This increased amplitude of vibration is transmitted to the basilar membrane, the hair cells and hence to the cochlear nerve. This is what we believe occurs by means of the artificial window. For it to be effective, the neural mechanism must be capable of transposing the sound waves into nerve impulses and transmitting them to the brain.

Briefly, then, those can be helped whose lesion prevents sound impulses from reaching the organ of Corti and whose cochlea and nerve are intact. How can these patients be selected?

Our first contact with the patient usually reveals his handicap unless he is especially adept at lip reading. We have no trouble in determining that his hearing is impaired. A simple lowering of the voice reveals it. Ordinary conversation in a quiet place is somewhere between 40 and 60 decibels above the threshold. With a slight effort it can be raised to 70 decibels above the threshold for the normal ear. If still the patient cannot hear, we can be fairly sure that his lesion is not of the pure conductive type, or rather that the neural mechanism is causing at least part of his deafness. This point is not absolutely proved, but from experiments conducted by Bunch and audiometric readings done on patients with as much of a conductive lesion as it is possible to produce, the thresholds are around 60 decibels above normal for speech frequencies. This may well be the explanation of the fact that many persons reach a so-called level, at which their hearing remains for many years; in other words, if the footplate of the stapes is completely fixed by a process such as otosclerosis, further deafness does not occur until the neural mechanism becomes involved. Then there may also be evidence of a perceptive lesion as frequently shown by decreased bone conduction or the loss of high note perception.

Continuing with further questioning of the patient in regard to his deafness, we are interested in the time of onset and the course which it has seemed to take. Has it progressed? Has it seemed to reach a level? Is the hearing normal at any time? The fistulas are known to be of service only to those whose hearing defect has started without other obvious ear diseases; that is, without ear infection. An accompanying head cold at the time of onset of the deafness is often mentioned but this is not considered as evidence of actual ear disease. Often the deafness is in one ear and may remain so for many years before the other one becomes noticeably involved. Little disability is herewith encountered and the patient usually does not seek advice until the better ear is noticeably affected. This, of course, means that the hearing in the better ear has been gradually diminishing but is not noticed until the threshold is somewhere between a 40 and 60 decibel loss for the critical frequencies.

We hear much about hereditary deafness, but in my group of patients in whom the lesion can be due to otosclerosis, less than half give evidence of familial disease of the same type. Such history is so inadequate and so vague that usually it is of no importance with any particular individual. Careful investigation of this problem is necessary and the present statistics are inadequate. If there are many young people in the same family who are deaf, there can be a strong

suspicion of otosclerosis; but this does not influence us either way when we are to decide about an operation.

The patient's previous treatment should be investigated. Usually something has been done, and its effect should be noted. If actual improvement can be conclusively demonstrated by any type of nonoperative treatment, serious consideration should be given to its continuation before operative therapy is advised. Here false hope is often engendered and a patient will report improvement if he has actually subjected himself to a particular type of treatment. Audiometric readings may be of help, with due consideration for their comparative reliability.

Tinnitus is a frequent but by no means a constant finding. Its variations are well known and it frequently disappears following the fistulization operation. Its presence constitutes a minor indication for operation but its absence is not a contraindication.

Vertigo is a very important symptom to investigate with great care. If it is present, a labyrinthine disturbance must be suspected until definitely ruled out. The labyrinthine and cochlear function are so often both involved that vertigo must be entirely eliminated before operation is advised.

Pain, so often confused with headaches and a dull feeling of fullness in the ear, requires a careful and very precise investigation. Especially those patients who have had head injuries and who have been unconscious, with residual dull aching anywhere in the head, should be suspected of some lesion which in itself may be causing the deafness. Severe headache, especially if prolonged, must now be considered definite contraindication to operation until it can be relieved. Actual pain in the ear itself must be eliminated before operation.

Previous diseases, especially the acute exanthemata, often usher in the deafness. If suppuration has not followed one of these diseases, the deafness is usually due to a lesion of the neural mechanism and will be detected during the functional examination of the ear.

A point in the anamnesis about which I always inquire concerns the influential factors. Does the patient's hearing improve or become worse under certain conditions? Fatigue, infections and cachetic states depress the hearing below the usual level for that person, and mild variations are frequently noted. The important point here is, does the hearing ever return to what the patient believes is normal,

and what are his criteria of normal? If the patient's hearing is hovering around the critical threshold for speech, minor incidents may change it from good hearing to poor. Thus, bodily fatigue, emotional upsets and upper respiratory infections depress hearing, and if avoided, may permit a somewhat longer period for careful analysis of the case before operation is advised, or other advice given.

Has a hearing aid been used, and with what result? Those who are satisfied with, or can be adjusted to a well-fitting hearing aid are fortunate indeed and for the present should not be urged to have an operation. They, of course, will prefer to hear without it and can use it later if the operation fails, but if the person is psychologically adjusted to the aid and has accepted his infirmity without damage to his psyche, he should be encouraged to continue until further improvement in the other types of treatment are forthcoming. Also, in regard to the use of the hearing aids, the patient's occupation and the time which he has available for the operation and the postoperative period must be considered. For anyone who falls within our rigid group of those whose hearing can be improved by operation, a properly fitted hearing aid will also be a great assistance and should be tried before operation. Adjustment to the instrument may be difficult, especially if the tinnitus is severe, and the patient may refuse even to try such a device. However, he must have the opportunity, because if operation fails, the instrument may be necessary later and the patient must have a knowledge of its effect before a questionable procedure is undertaken. In this the utmost cooperation is necessary between the hearing aid representative, the doctor and the patient. So important is the proper fitting, that no one instrument can have had a fair trial until an individual ear piece properly made for the particular person has been used. If a patient must, therefore, be at work without interruption, a hearing aid is preferable to operation and he is entitled to information concerning this possibility.

Some drugs depress the hearing and effort is now being made to determine the relationship of hearing loss to anesthetics and labor inducing drugs administered to a mother at the time of delivery. Habitual users of any drug, even the common antipyretics and sedatives, should have the benefit of a period of abstinence before operation, especially if there is any evidence of neural mechanism damage by the functional test.

The foregoing constitute the specific points which must be considered during our preliminary contact with the patient. There are still much more important features to be considered which I shall

call the psychological aspects of the particular case. They are difficult to classify and frequently defy analysis, but as we continue to talk to our deafened patients, we gradually become aware of their life situations and the importance of better hearing for their proper social adjustment. The great fear which most deafened people have is of further loss, or possibly complete loss, of the hearing function. This so seldom occurs that some people will be well pleased with no more than the assurance that the chances are very good that no further loss will occur. Advice to the patient which will preserve his general good health may be all that that individual requires. We have all seen very adequately adjusted deafened people who would not, under any circumstances, wish to have their hearing improved. The number of these is unknown because they usually do not ask advice, but I do know that they exist and occasionally seek consultation on the pleas of relatives and friends.

Something of the environment in which the person lives should be noted during the interview. Does his handicap inconvenience other people with whom he comes in contact? These and many other questions will come to mind as we spend more time with our patients. The central aim of our medical advice to any particular person is to re-establish a satisfactory adjustment to the environment in which he finds it necessary to live.

In the last analysis the final test of our advice and treatment will be the degree to which the patient is readjusted to his particular social situation.

To summarize the important points in the anamnesis, we include a history of noticeable deafness for at least six months with or without tinnitus and without periods of apparent normal hearing. This period is, of course, always preceded by a time during which the patient is unaware that his auditory acuity is abnormal. Anamnestic contraindications are previous purulent otorrhea, attacks of labyrinthine vertigo, pain around the ear or prolonged headache, satisfaction with an artificial hearing aid or a psychologically difficult or socially unfortunate patient who cannot for some reason take the time for proper preoperative and postoperative care.

It is assumed that all general considerations for a delicate operation have been evaluated. Good general health, absence of acute infection or chronic disease, such as syphilis, tuberculosis or blood dyscrasias are prerequisites. Abnormal blood pressure, allergic states, endocrine disorders and nutritional deficiency must all be adjusted before operation is advised.

With the history as we have obtained it, what more is necessary before the patient can be advised to have the fistulization operation? The roentgenogram of the temporal bone will show the extent of the pneumatization. The external horizontal semicircular canal can be demonstrated. Sclerotic and previously infected mastoids are a contraindication to operation at present, although, of course, there can be patients who have had mastoid infection and sclerosis of the bone and obliteration of many of the air cells without permanent cicatricial damage to the middle ear. However, since the hearing loss can be due to fixation of the stapes footplate or to scar around the round window, or both, definite evidence of previous infection in the middle ear must remain a relative but not an absolute contraindication. At some later date when it is possible to determine whether or not the round window niche is scarred, we may be able to include some of the previously infected ears in the operable group.

The nose and throat must be in good condition. Nasal obstructions which are clinically symptomatic and chronic infection in the pharynx and tonsils should receive consideration before operation. Any evidence of skin irritation of the pinna or surrounding structures should be completely eliminated. The external auditory canal must be clean and open to provide a good view of the tympanic membrane.

With the pressure speculum of the Siegle type, movement of the tympanic membrane can be noticed. Sometimes the malleus handle can be seen to move, but even if it cannot, no conclusion regarding the functional activity of the ossicular chain can be drawn therefrom. The actual movement necessary to stimulate the inner ear is so infinitesimal that gross observation with the naked eye can give no information about the functional movement. Many normal ears have no visibile movements of the ossicles as seen with the naked eye. Movements of the membrane itself, however, may demonstrate adhesions within the tympanic cavity. These are evidence of previous otitis media.

The eustachian tubes should be examined and simple insufflation of air well demonstrate their patency. The pharyngeal end of the tubes should be seen by means of a nasopharyngoscope or a postnasal mirror, and when normal I have yet to find a tube through which air cannot be passed to the middle ear as shown by the patient-to-examiner tube placed in the external meatuses.

This concludes the necessary features of the objective examination so that we now turn our attention to the functional impair-

ment as shown by various tests. Determinations of quantitative loss can be made with calibrated tuning forks if carefully enough performed. At least four forks should be used for air conduction tests between 128 and 4096 cycles, and if the loss is between 40 to 60 decibels for these frequencies, the lesion can well be a fixation of the footplate of the stapes. Bone conduction tests with tuning forks are unreliable, but if the patient can hear the 256 and 512 forks as long or longer than the examiner in a quiet but not soundproof room, the neural mechanism may be intact. Tuning fork bone conduction tests above 512 are so interfered with by air conduction from the same fork at the same time that they are not accurate. It is, therefore, impossible without some type of bone conduction electric receiver to get a relatively clear idea of the bone conduction above 512 cycles. Therefore, a final functional examination cannot be obtained by tuning forks alone, and the audiometer must be used.

The functional examination of the vestibular portion of the inner ear is easily done by the caloric method. Fifty c.c. of water at 50 degrees when applied against the tympanic membrane will stimulate the normal labyrinth as shown by induced nystagmus, and a feeling of nausea and vertigo on the part of the patient. If the labyrinth cannot be stimulated by the caloric method, serious consideration must be given to the possibility of a neural mechanism change which causes the deafness, in which case the fistulization operation would be of no benefit to the patient.

The following is still controversial in some details and accurate interpretation is still often impossible. Until further standardization of the instrument is forthcoming, discrepancies will occur. However, we can say that a patient whose average of repeated audiograms indicates an air conduction loss of 40 to 65 decibels in all frequencies between 128 and 8192 cycles, whose bone conduction is approximately normal in the same frequencies and whose high limit is not below 12,000 cycles per second, will be improved with a fistula, providing the patient presents the other requirements as mentioned above. From this, certain deviations probably will not contraindicate operation, but what deviations should be allowed, we do not know with certainty.

As time goes on it may be possible to include other people in the group who can be helped by the operation. Lempert[4] has found improvement in a number of people who were considered to be unsatisfactory before the operation was performed. In just what respect they were unsuitable has not been clearly stated.

I wish to present the findings of a typical case which has been improved by the fistulization operation and then I will demonstrate the audiometric findings on two patients for whom the fistula has been of no benefit. The reason for this, I believe, can be logically explained, although as yet it has not been definitely proven.

A CASE OF FISTULIZATION FOLLOWED BY IMPROVEMENT

This patient was a 25-year-old female bookkeeper who fulfilled all the requirements mentioned above. She had been noticeably deaf for about two and one-half years. One sister is somewhat deaf in one ear. Treatment for the deafness has consisted of eustachian tube inflations and nasal sprays. She had tried an electric hearing aid but was not satisfied with its use. She had taken no harmful drugs during her lifetime. Psychiatric examination by Dr. Gates revealed that "she feels out of things" and that "her attitude toward her deafness is a fairly satisfactory one. She is sensitive, and with increasing deafness it is quite possible that she might become somewhat paranoid."

Physical examination revealed that the tonsils had been removed, there was a slight septal deviation without evidence of chronic infection. The right ear was normal in all respects to objective examination, with an open eustachian tube, increased bone conduction by a 256 fork in a quiet room, negative Rinné and audiometric findings as noted in the accompanying chart.

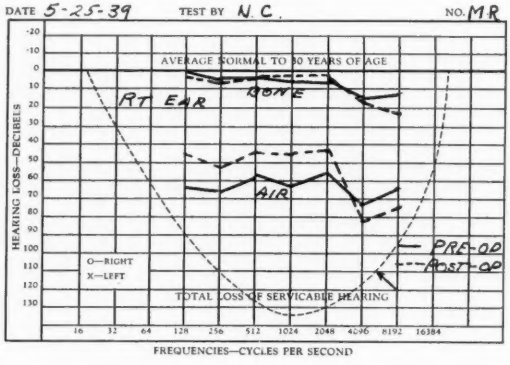


Fig. 1. Preoperative and postoperative audiometric levels of a patient who obtained noticeable improvement following fistulization of the otic capsule.

In all the audiometric examinations the left ear was slightly worse than the right, but because of scars on the tympanic membrane of the left ear, it was thought that she had probably had infections in that middle ear in her previous life. Consequently, the right ear was selected for operation and it was performed on May 25, 1939.

We note from the audiogram that the hearing for the critical frequencies of speech, range about 60 decibels loss throughout the seven octaves. This makes it necessary for a person's voice to be slightly raised for her to understand speech. This was found to be the case, and with the voice slightly raised, she could hear fairly well. However, it was impossible for her to hear the telephone in the office, and much of the conversation which she wished to hear was inaudible.

Thirty days after operation the hearing improvement by audiometric tests was noted. This improvement was such that she could hear much more conversation than before operation. It is apparent that her hearing now is just within the conversational range but subjectively she feels that her hearing is tremendously improved. Her comment to the psychiatrist was "it is much better than I expected."

A CASE NOT FOLLOWED BY IMPROVEMENT

Many people have wondered why would not hearing loss due to a long-standing otitis media be improved by operation if the hearing improvement depended simply on the presence of a new fistula in the external canal. To examine this I operated on a patient who had had a long-standing chronic suppurative otitis media for about twenty years. The patient was a 28-year-old woman on whom I had performed a radical mastoidectomy in August, 1938. The cavity healed successfully and was completely dry and epithelialized about four months after operation. The healing process provided a well-lined cavity in which the external wall of the horizontal semicircular canal was easily identified through an enlarged external auditory meatus. A high-pitched tinnitus was extremely loud in this particular patient, which was one of the inducements to operation. This patient also had a chronic suppurative otitis media on the left side, so that the hearing loss on both sides was approximately the same. The hearing loss in this patient was considered to be due to a fixation of the otic capsule windows but was not thought to be due to otosclerosis.

On February 1, 1939, a fistula was made in the right external horizontal semicircular canal. There was no subjective improvement

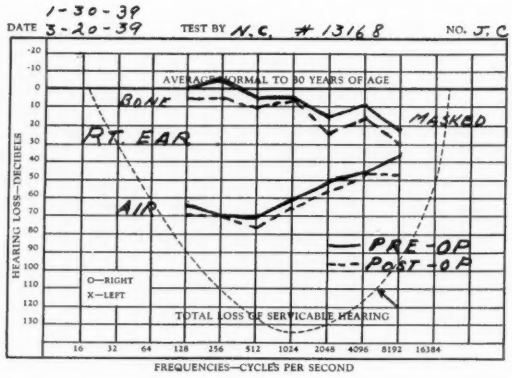


Fig. 2. Preoperative and postoperative audiometric levels of a patient who had healed radical mastoidectomy before fistulization of the otic capsule. No improvement.

in hearing. The tinnitus changed in character somewhat but did not completely disappear. Preoperative audiometric examination is designated on the accompanying chart. Thirty days after operation there was no demonstrable improvement in hearing.

If we accept the present belief that the fistula can only be of service to those patients in whom the round window also functions, it can be assumed in this case that the scarring in the middle ear had affected the resilience of the round window as well as the oval window.

The second case I wish to mention briefly, which did not respond to the fistulization operation, was in a 23-year-old girl who presented a history of deafness with many features which fall within our rigid group, but who also presented several other findings which we now consider contraindications to the operation.

The history was rather typical of a progressive deafness in both ears, but as well as the deafness, the patient complained of severe and contant headaches for which nothing she had ever done gave real relief. Careful psychiatric examinations of this patient were made on several occasions and accurate evaluation of the headaches was not very definite. Examination of the ears by inspection revealed no abnormalities and the patient stated that at some previous

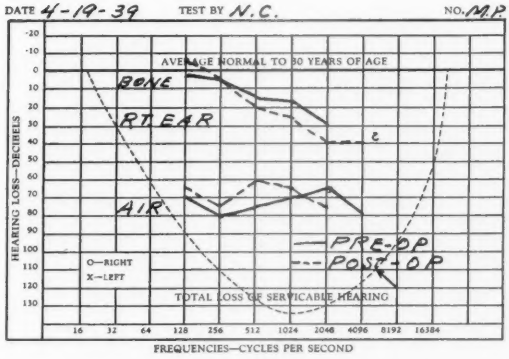


Fig. 3. Preoperative and postoperative audiometric levels of a patient who obtained no noticeable improvement after fistulization of the otic capsule.

time she might possibly have had an otitis media, but this was not definite. X-rays of the mastoids showed no evidence of abnormal pneumatization. Examination of this patient's audiogram revealed a loss by air conduction, as shown, and by bone conduction in the lower five frequencies, possibly a little subnormal. It should be noted, however, that for the frequencies of 4096 and 8192 the bone conduction receiver was not heard at the loudest intensity possible with our audiometer. However, since these two frequencies are not absolutely necessary for speech, it was thought that possibly with good bone conduction for the lower five frequencies, air conduction might possibly have been improved so that speech could have been heard somewhat better.

A fistula was made in the external horizontal semicircular canal on the right side on April 17, 1939, at which time there was no subjective improvement in hearing on the operating table. In the days which followed, the ear healed satisfactorily with no change in the headache. Hearing improvement did not occur at any time during the postoperative course.

Further investigation of the headaches involved the administration of many types of drugs and pneumo-encephalography. Cortical brain atrophy was diagnosed by roentgenography. The headaches were not relieved. The operation had no effect on the hearing, the

tinnitus, which was slight in this case, or the headaches, so that I now believe that such audiometric findings and severe and long-standing headaches should be definitely considered as contraindications to the fistulization operation.

Further functional testing of the hearing can give us additional information but it is not absolutely necessary at the present time when we are deciding whether or not to advise the fistulization operation. From the research standpoint, however, testing must be improved and intelligibility tests will certainly be added as time goes on. They give much promise for the future, and when they can be carefully enough standardized, they will, of course, add considerably to our final analysis. They will be of particular importance in evaluating the degree of improvement.

The loudness balance test of Fowler probably gives positive differentiation between a perceptive and obstructive lesion, provided that for the lower frequencies there is a threshold difference of at least 15 decibels. Since most of the patients who fall into our group, as noted above, do not have this difference, this test is not of much value in deciding whether or not to operate.

The value of repeated audiograms and the possible variations over a long period of time have been carefully demonstrated by Witting[5] and other authors, so that in our final interpretation before operation is advised, at least three audiograms must be made before an accurate preoperative level can be established.

SUMMARY

The overwhelming evidence now is that after a fistula is made through the otic capsule of partially deafened persons, the internal ear has been rearranged so that sound of a certain intensity can stimulate the organ of hearing, whereas before the fistula was made, sounds of the same intensity were inaudible. From the reports which have been presented, I consider that the cases which have the best chance of improvement are those in which the sound waves are obstructed in their passage to the organ of Corti where this structure and the remainder of the neural mechanism stay normally intact. Some of the reports seem to indicate that ears which may have a defect in the neural mechanism have also been improved by fistulization, but these cases require further experimental study before they can be placed in the predictable group of those in whom improvement can be expected.

789 Howard St.

BIBLIOGRAPHY

1. Jenkins, C. L.: Otosclerosis. Tr. Internat. Cong. Med. Sec. 16 (Otol.), 2:609, 1913.

2. Bárány, R., Quoted by Fremel, F.: Demonstration eines wegen Otosclerose vor 8 Jahren operierten Patienten. Monatschr. f. Ohrenh., 56:552, 1922.

3. Holmgren, G.: The Surgery of Otosclerosis. Nelson's Loose Leaf Surgery of the Ear, 1938.

4. Lempert, J.: Improvement in Hearing in Cases of Otosclerosis. Arch. Otolaryng., 28:42-97 (July), 1938.

5. Witting, E. G.: A Brief Survey of Audiometry. Abington Memorial Hospital, Abington, Pennsylvania, 1939. (Unpublished.)

XXI

THE TYPE OF DEAFNESS WHICH RESPONDS TO FISTULIZATION OF THE OTIC CAPSULE

NORTON CANFIELD, M.D.

DISCUSSION BY

WALTER HUGHSON, M.D.

For the past two years every otologist in the country has become keenly alive to the possibilities of this relatively new surgery of deafness. The whole problem is unique in several of its many intriguing aspects. In the first place, deafness as such is never a condition which demands emergency treatment. The very fact that patients defer adoption of any radical measures until the hearing loss has usually become profound, is ample proof of this statement. After a relatively static level has been reached in the hearing loss, operation can be postponed almost indefinitely without seriously jeopardizing the eventual outcome of the surgical measure employed. A statement often made in support of surgery, and usually with little basis in fact, is that whether improvement is obtained or not, the hearing will be maintained at its present level as a result of the therapy. Only when data becomes available as to the effect of surgery in early hearing losses, can this inducement be advanced with any degree of accuracy. At the moment the prognosis of deafness following any form of treatment is a matter of great uncertainty.

Under the circumstances, and since surgery is purely a matter of election, such carefully measured standards of selection, as Dr. Canfield has just presented, are entirely requisite and most reassuring. There can be no question that the future success of any radical treatment of deafness will depend upon such meticulous care in the choice of patients for operation.

There are certain points in Dr. Canfield's discussion which I shall ask him to elucidate and certain questions which arise in relation to his exposition of the rationale of the fistulization operation.

Can the inner ear properly be considered a rigid box, as Dr. Canfield has described it, unless both windows are fixed and the possible routes of fluid exchange to and from the scalae definitely proved nonfunctioning? Reference is also made to the necessity of having

a freely movable round window membrane. Preoperative determination of this fact would probably be difficult. In a series of cases in which exploration of the middle ear was performed for examination of the round window niche, entirely unexpected adhesions have been found on numerous occasions and repeated observation has shown that fixation of the round window membrane does not further impair a conductive deafness already present. Guild showed in 1930 that middle ears which had once been the site of suppuration, with resultant fixation of the round window membrane by fibrous tissue, showed less impairment of hearing than those in which the niche remained uninvolved. Even though there has been no history of acute suppurative middle ear disease, there may still be found some pathologic reaction to an infection which has never reached a symptomatic stage and which has left little, if any, gross evidence of its presence, except in an insignificant retraction and thickening of the drum membrane.

Although Dr. Canfield and others have referred to the patency of the eustachian tube, it is not quite clear whether this is a requisite of operability or not, and, if so, why its closure should influence adversely, the effectiveness of a new fenestrum. The actual diagnosis of deafness is often a matter of great difficulty but if all infections of the ear, which have gone on to suppuration or not, are to be considered as contraindication to this surgery, is it meant that every operable case exhibits impaired mobility of the ossicular chain or fixation of the stapes footplate as a result of an otosclerotic process? We have not felt that the diagnosis of otosclerosis could be arrived at with quite so definite a degree of finality.

Dr. Canfield has discussed the hearing level in relation to operability but has not mentioned what improvement may be expected in favorable cases. With a margin of 30 decibels, 40 to 70 decibels loss is the operable range will the patient with the more profound impairment have a reasonable chance of regaining useful hearing?

In connection with the air conduction level, impairment in bone conduction and the implication of neural damage has been discussed. The operation can be considered only in the presence of a completely intact neural mechanism. On several occasions, we have drawn attention to the fact that bone conduction may be affected adversely by many factors other than actual nerve atrophy. It is further felt that true nerve deafness can only be demonstrated conclusively when both the loudness balance and bone conduction tests are indicative of it. One of the most obscure points in the selection of operable cases is this requirement of an intact neural mechanism.

We know, for instance, that old age or neural deafness can often be compensated for by an appropriate hearing aid. Why, then, does the increased perception of loudness resulting from the fenestration not serve for some of the higher frequencies as well as the low? Certainly impaired bone conduction cannot be accepted as evidence of complete neural destruction. I realize that this standard is set as a result of actual clinical experience, but the reason for it is far from clear.

Dr. Canfield's clinical criteria can scarcely be questioned. It would seem, however, that a few restrictions were laid which, if followed completely, might limit the number of cases selected for operation unnecessarily. The use of drugs and drug addiction are quite separate things. A hearing loss due to drugs would surely show some inner ear involvement. The mere fact that drugs are used in moderation or even to excess, in itself, is no bar to surgery. Individual susceptibility to the effect of drugs is the critical factor. Were this restriction carried to a logical conclusion, alcohol would be one of the first things to consider, and yet its association with deafness is certainly not given undue importance.

It is common practice, and undoubtedly properly so, to require a negative blood Wassermann before recommending any elective type of surgery. However, direct evidence that lues is an etiologic factor in the development of deafness, is, according to Ciocco, an extremely unusual finding. Here again, were the lues a factor, the lesion would be neural and the case automatically inappropriate for fistulization.

The very obvious conservatism manifest in this presentation of Dr. Canfield's is wholly admirable. In this discussion, an attempt has been made to break down certain apparently unnecessary barriers which he has raised to surgery. However, his position is a completely sound one. His careful psychologic study of his patients is of the utmost importance. Although not emphasized in his paper, the completely unreasonable early subjective reaction to any form of therapy must be a matter of no importance in the final analysis of the effect of the surgery of deafness. A friendly word by the physician with the inevitable enthusiastic response from the patient is no measure of successful therapy. Only by often repeated audiograms, careful appraisal of speech intelligibility and ultimate acceptance of subjective improvements, as exhibited by improved social and economic relations, can we feel that any particular surgical procedure has been of the slightest practical value.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, October 2, 1939

THE PRESIDENT, DR. GEORGE T. JORDAN, IN THE CHAIR

Pathology of the Inner Ear in a Case of Deafness From Epidemic Cerebrospinal Meningitis

E. W. HAGENS, M.D.

(This paper is printed in full on page 168 of this issue.)

DISCUSSION

DR. JOHN R. LINDSAY: The sections which Dr. Hagens showed are excellent illustrations of the changes which occur in the labyrinths when there is invasion from the meninges. Temporal bones from such cases are not easily found and therefore reports in American literature have been scarce or absent.

I have had the opportunity of examining several cases of Prof. Nager's collection in which the meningitis had occurred many years before death and in most of these the labyrinth spaces had been completely obliterated by new bone formation. The question comes up, then, as to whether the case described by Dr. Hagens, if it had survived, would have gone on to extensive ossification and obliteration of the labyrinth spaces with complete loss of function, or whether this might have been one of the cases with some vestiges of function remaining.

Up until recent years the meningococcus has been responsible for most cases of total or subtotal loss of hearing due to meningitis. Now that some cures of streptococcus and pneumococcus meningitis are being obtained some cases of total deafness are likely to appear among these cases. I had the privilege of examining one such case last year.

We have good reason to believe that the labyrinthitis from meningococcic meningitis behaves in essentially the same way as

labyrinthitis from streptococcus or pneumococcus meningitis, and of these I have more than sixty bones in my collection. I have selected a few slides to show some of the very early steps in the invasion of the labyrinth from the meninges, and also some of the later findings in cases of labyrinthitis of long standing.

(Slides 1 and 2) Collection of pus at mouth of aqueductus cochlearis; (Slide 3) Invasion of scala tympani through the modiolus; (Slide 4) Healed labyrinthitis—showing new bone, and connective tissue in the scala vestibuli and scala tympani and marked dilatation of the ductus cochlearis; (Slide 5) Section through basal coil near round window showing bone overlying the mouth of the aqueductus cochlearis.

Dr. Alfred Lewy: I would like to report a case which was somewhat parallel to that of Dr. Hagens, although we do not have the histologic evidence. This concerned a female child, aged 7, who entered County Hospital with a frank meningitis from which the hemolytic streptococcus was cultured on three or four occasions, following a right-sided otitis media of about two weeks' duration. The left ear was totally deaf with no response to caloric stimulation. Dr. Leshin did a simple mastoidectomy, uncovering the dura on the right side. On the left side a radical was done with no signs of infection of either the middle ear or mastoid cells. As she had had good hearing prior to this we believed the destruction of hearing in the left ear was due to involvement of the inner ear, or the nerve, by meningitis originating from the right ear due to streptococcus hemolyticus. She recovered, but for months afterward was unable to walk steadily in the dark. That is about two years ago, and now she is still showing some mental disability, is easily fatigued and able to attend school only for part time.

Dr. Frank Novak: I would like to ask Dr. Hagens whether the cochlear involvement preceded the vestibular.

Dr. Howard Ballenger: I would like to ask Dr. Hagens whether, when he looked up the literature, he found the pathology in any of these cases a meningoneuritis rather than a labyrinthitis. We see frequently in infectious conditions such as mumps, scarlet fever, etc., with or without a middle ear involvement, what is presumed to be a meningoneuritis, rather than a septic labyrinthitis. These neuritis cases would be more likely to have a marked partial deafness than the total type. The static branch to the labyrinth may or may not be involved. Some cases of marked deafness in meningococcic meningitis may be this type. Such reports as Dr. Hagens has just made will be the only way to settle the question.

DR. ELMER W. HAGENS (closing): I wish to thank Dr. Lindsay for his discussion, and certainly enjoyed his slides. I cannot answer Dr. Novak's question—I could not tell whether the cochlea was involved first. I could not tell from the sections and did not have the history. I never saw the child and was given what history there was by the resident.

With reference to Dr. Ballenger's question, I have always wondered what the pathology was in these cases. I have read it, but have never seen such a case. When we examined the 5,000 children I wondered just what the pathology was, and determined that if we ever got a case I would follow it up and see what the labyrinths showed. In this case there is destruction of the organ of Corti and loss of spiral ganglion cells in one ear, and partial loss in the other. Scarpa's ganglion cells were intact in both ears. I did not look up the literature completely. I do not recall a statement that the pathology was neuritis only. As a rule neurolabyrinthitis is mentioned and I think from the case I have and from what I found in Politzer that this is true.

The Surgical Treatment of Cancer of the Larynx

CHEVALIER L. JACKSON, M.D.

(Abstract)

Surgical treatment has the best statistical results to show in the treatment of cancer of the larynx, but in certain cases the patient may be permitted a choice or the laryngologist may feel justified in advising irradiation. The ultimate prognosis will be best in the better differentiated slower growing, lower grade tumors, regardless of the method of treatment. Grading should be taken into consideration in selection of treatment particularly in borderline cases; and the less differentiated higher grade tumors should incline one toward more radical surgery, or irradiation, although in these cases prognosis is poor by any method of treatment.

Surgical Indications: 1. Lesions occupying the middle third of one vocal cord are suitable for the operation of laryngofissure by the clipping technic, regardless of histologic character or grading. 2. Lesions reaching the anterior commissure and even involving the opposite cord are amenable to extirpation by the laryngofissure route also, but in such cases the Chevalier Jackson anterior commissure technic should be used. 3. Lesions in which the growth is cordal, but has reached the posterior end of the cord and produced impair-

ment of motility, or has extended subglotically, call for total laryngectomy.

Laryngectomy and pharyngotomy are also done in cases where the lesion is extrinsic but the results in these cases are very unsatisfactory and it is possible that irradiation would accomplish as much in arresting the growth and prolonging life. On the other hand in some of the extensive cases, especially in those with cartilage involvement or perichondritis, there may be some advantage in removing the larynx preliminary to irradiation.

The indications for and technic of laryngofissure and laryngectomy as carried out at Temple University Clinic were given in detail and the essayist concluded his paper with a discussion of voice and speech instruction following operations on the larynx.

DISCUSSION

DR. PAUL HOLINGER: We are highly honored in having so distinguished an essayist start the new season. I know we all appreciate the vast experience that has given us the significant conclusions which Dr. Jackson presented this evening on this highly controversial subject.

I was glad Dr. Jackson stressed the necessity of biopsy in spite of the fact that his paper dealt only with treatment. Certainly the grading of the lesion is an extremely important factor in the choice of therapy, to say nothing of its importance in establishing an accurate diagnosis.

The extremely interesting question of voice following laryngeal surgery was beautifully illustrated this evening with the sound motion picture film. There is a great danger, however, in attempting to rationalize in the choice of a surgical procedure in an effort to give the patient a good voice. This is usually only wishful thinking, dangerous not only to the patient but also to the final statistical analysis of the merits of the various procedures. If we could adhere strictly to the indications Dr. Jackson stressed, possibly our series of permanent cures could approach the 85 per cent he reported in suitable cases.

One question I should like to ask deals with work being carried out at Temple University by Drs. Fay and Smith. Has any experimental refrigeration work been carried out to relieve the pain in far-advanced extrinsic carcinomatous lesions of the larynx?

I have enjoyed the paper very much and wish to thank Dr. Jackson again for coming to start the year for us.

Dr. Francis Lederer: A year ago we opened our season with a similar discussion of cancer of the larynx and at that time I had occasion to speak about rehabilitation of the voice and showed a number of examples. I have not been so fortunate as Dr. Jackson in getting the perfect voice he demonstrated on his patients following laryngofissure. Perhaps my patients have not used the ventricular bands as much as his have. It is a startling and beautiful result.

I have always been interested in the rehabilitation of the voice following laryngectomy, because it would be a severe jolt to a patient if you could not promise any voice. On previous occasions we have had the opportunity to observe patients who have developed a bucco-esophageal voice following laryngectomy by their own efforts, and we have seen the value of instruments that are made to give the patient an artificial voice. In introducing what I wish to demonstrate to you tonight, I want to introduce it apologetically, because I feel personally grateful to the Western Electric Company for their efforts in making the artificial larynx, having in mind that patients have not universally responded very well to it. I wish to demonstrate an apparatus which Dr. Jackson has seen before, by the originator of the instrument, who himself is a laryngectomized patient, and this individual knows what a voice should sound like. By his efforts and those of Dr. Hanson, of East St. Louis, Illinois, a device has been worked out which is the best I know of. It takes away the metallic sound which characterized the other instruments. The other instruments have not allowed for modulation of the voice as this one does. So I have asked the same lady who spoke to us last year to demonstrate the instrument which she introduced me to.

(The patient, Mrs. B., stated that her present apparatus (artificial larynx of Roberts-Hanson) has produced an effortless modulated tone. She finds that whereas other types of artificial larynges made necessary a very concerted action of the diaphragm this particular apparatus could be learned with greater ease. While she was particularly grateful for the previous instruments that have been produced, the apparatus which she now possesses is one that has made her very happy.)

Dr. Thomas C. Galloway: I think we are very fortunate to have Dr. Jackson demonstrate again that by a relatively simple procedure in properly selected cases the patient with laryngeal cancer has 85 per cent chance of cure with a useful voice and a normal air-

way, and a chance for a normal life with little risk to himself. I think it is too bad that this information could not be more widely disseminated, because in Chicago the feeling has become widespread that irradiation is the treatment of choice for such a carcinoma. Certain channels of propaganda are closed to such a presentation as Dr. Jackson's like advertising in local medical publications of a device which was demonstrated here last year, which might have possibilities and has been useful at least in one case. Such advertising may imply to the general practitioner that such a method allows the cure of cancer with no risk to the patient, no pain and no distress, though there is little evidence to support such implication.

I think we all agree with all that Dr. Jackson has said, especially that in proper cases with no fixation of the cord, in early cancer in the anterior half of the vocal cord, laryngofissure is the treatment of choice. I do not want to depreciate the value of irradiation in many conditions, but I do not think that the impression should go abroad that at present anything can replace laryngofissure as the most satisfactory procedure in properly selected early cases.

DR. GEORGE E. SHAMBAUGH, JR.: Very little can be added to this complete and beautiful demonstration. I think Dr. Jackson should receive particular credit for introducing the dissection of the anterior commissure which allows more patients to be cured by laryngofissure. I have used this procedure in two cases, one of which you saw. One item in technic was the use of surgical diathermy cutting current in removing the cord after exposing the larynx, except for the vocal process of the arytenoid which has to be cut off by scissors. The removal was done entirely bloodlessly and very quickly and easily.

DR. THEODORE WACHOWSKI: I arise not to discuss Dr. Jackson's paper, but I would like to comment on what Dr. Galloway mentioned in regard to so-called advertising. I would like to say that the Roentgen Society has recognized these facts, and is trying to have this type of advertising eliminated. If your sentiments are made known, I think the Chicago Medical Society will try to permit only very ethical advertising in the Bulletin. I think the things Dr. Galloway referred to should not be permitted, and will not be.

DR. L. B. BERNHEIMER: If I understood correctly, Dr. Jackson said that in intrinsic, infiltrating lesions of the larynx the patient is given his choice between total laryngectomy and irradiation. I would like to know what Dr. Jackson's end results have been when radiotherapy was employed for this particular type of lesion. I ask

this question because I am associated with an institution where a large number of laryngeal lesions are seen, and where irradiation is used extensively. It has been our experience that these infiltrating lesions resulting in cordal fixation never respond to irradiation. This is one type of lesion where we do insist on surgical treatment, that is, laryngectomy. I cannot recall a single five-year cure of an infiltrating laryngeal lesion treated by irradiation.

DR. L. Z. FISHMAN: I would like to ask Dr. Jackson how many of the cases of intrinsic carcinoma of the larynx, were anaplastic and how many of the slow growing type.

DR. ADOLPH HARTUNG: This discussion did not deal with the relative value of irradiation and surgery, and I have therefore little to add. Certainly our results with irradiation have not been very satisfactory so far as the ultimate outcome is concerned. I think that surgery still has a place of choice except in special instances in the treatment of early lesions of the larynx.

DR. CHEVALIER L. JACKSON (closing): I appreciate the contributions of all those who joined in the discussion. Dr. Holinger asked regarding refrigeration. I have been asked about that before, but I cannot say anything about it because, so far, we have not contributed any patients on whom Dr. Fay and Dr. Smith have made these experiments. I say experiments, because that is the basis on which patients are still being taken for that work. That is clearly understood by the physician and patient—this refrigeration is a purely experimental thing as yet, and nothing can be promised. So far we have not referred any patients from our service. However, they are continuing their studies and will report further on them.

I was much interested in Dr. Lederer's presentation and the beautiful demonstration of the apparatus given by his patient. Certainly the mechanical larynx is an attractive field for continued and further work. It is our practice to give our patients postoperatively a systematic course of instruction first, to give them an opportunity to develop bucco-esophageal voice, which can be developed by many patients, more easily by men than by women. The work which in this country was started by Morrison of New York, is certainly productive of a better voice without apparatus than most patients will obtain without systematic instruction. On the other hand, if a preliminary period of training without apparatus is given, the next thing is to get the patient in touch with one of the better forms of apparatus, and it is from the patients themselves that most of the important improvements in apparatus have come.

I thank Dr. Galloway for his emphasis on some of the points in connection with laryngofissure. In Dr. Babcock's department, he uses steel wire in all sorts of cases, neck surgery and other cases, and we took it over for buried sutures in the neck and have found it extremely satisfactory. The chief point in its favor is that experimental work has shown that it is singularly free from irritating reaction in the tissues. When the sutures are superficial they may be felt under the skin and occasionally will be annoying and have to be removed, but if deep enough they cause no irritation whatever. We have been using them long enough to be well satisfied. On the other hand, I do not wish to lay any great stress on that point. Doubtless other materials are just as good, but we have used it enough to feel that we want to continue it. One point is that it is considerably stronger than silver. There are several different strengths, but in the size we use it almost never breaks.

I wish to congratulate Dr. Shambaugh on the beautiful result in the patient we saw this evening. I think the voice will still improve. We have found that the improvement continues for a number of years. I think it unwise to force them. It should be cultivated in quiet places and not forced in noisy places. I think this patient will get better improvement, although the voice may be higher pitched than normal because of the shortness of the cords. It is a very symmetrical larynx with no granulomatous approximation and I think it will improve. One patient that we have, six or seven years after laryngofissure, claims that every six months she can make a comparison with her friends and be assured that she is not wrong in her own conviction that her voice carries further and is still improving.

Dr. Wachowski's remarks do not call for a comment from me, as I am not familiar with the matters concerned. In regard to Dr. Bernheimer's point, which is very important, we do not give the patient the choice. I said that in cases not suitable for laryngofissure for one reason or another, we take the patient into consideration and go over the problem with him and his family, but not in cases where there is impairment of motility. I do not say they are necessarily good ones for irradiation. If laryngectomy is not done, some treatment must be given. I have not the figures at hand as to what percentage were anaplastic. I know this is a great minority.

Abstracts of Current Articles

NOSE

Nasal Syndrome in Cranio-Facial Leontiasis. (Sindrome nasale nella leontiasi cranio facciale.)

Coppo, E. (Roma), Valsalva, 14:76-91 (Feb.), 1938.

This is a rather complete literary review of the etiology, pathological anatomy and the clinical picture of this disease. Coppo reports an exceptionally interesting case of leontiasis in which excision of the right superior maxilla was performed in toto, due to severe symptoms, uncontrollable trigeminal neuralgia, and the youth of the patient. A prosthesis was employed postoperatively with very satisfactory clinical and cosmetic results.

Resection of this region was never before employed for the relief of this malady and therefore worth recording.

SCIARRETTA.

PHARYNX

Plastic Operation of the Palate Following Removal of Large Osteoma of the Superior Maxilla. (Di un metodo personale di plastica del palato dopo asportazione di voluminosi osteomi del mascellare superiore.)

Pietrantoni, L. (Brescia), Valsalva, 15:205 (May), 1939.

Extensive mutilating operations on the superior maxilla for removal of benign tumors have caused Pietrantoni to use various types of plastic reconstruction of the palate, yet these have failed in most instances to give satisfactory results. He, therefore, has successfully used a new, original method.

The operation is performed by the usual external median incision and the maxilla removed with the tumor. The steps of his technic follow.

First, the nasal mucosa is elevated from the naso-antral bony wall from the floor to the superior boundaries of the middle meatus, and as far back as the choana, and then incised along the upper margins, leaving it attached to the floor.

Second, the bony wall is removed completely, taking special care that it is left perfectly smooth at the floor.

Third, the mucosa of the cheek is separated a few centimeters from the underlying tissue at the line of incision.

Fourth, this buccal mucosa is fixed to the fibromucosa of the hard palate by interrupted sutures. When this suturing is completed, the nasal mucosa is drawn over the raw surface of this newly constructed palate.

Pietrantoni has never considered it necessary to suture these two layers together, but he holds them in contact with lightly packed gauze strips which are removed through the nasal cavity after the fifth or sixth day. Finally, he completes the operation by suturing the external median incision.

The article is illustrated by four colored tables describing the operation and various photographs of the patients, tumors and results obtained.

SCIARRETTA.

Presence of Cartilage Nodules Located in the Submucosa at the Base of the Tongue. (Sulla presenza di noduli cartilaginei sottomocosi in corrispondenza della radice della lingua.)

Bachi, S. (Torino), Valsalva, 15:122 (March), 1939.

The writer reports two cases in which a small nodule of hyaline cartilage surrounded by a strong fibrous capsule was found in the tongue near its base.

Bachi discusses the various theories of the genesis of cartilaginous or bony inclusions found in the palatine tonsils.

He believes that these hyaline cartilages are embryonal residues separated from the second branchial arch. This conclusion is based first, on the fact that one specimen was from a child three days old, and therefore would exclude a metaplasia; second, because cartilaginous inclusions were found in the tonsil and the submucosa at the base of the tongue of the other patient, fifteen years old.

He further excludes the possibility of the cartilage originating from the lingual septum which was previously reported in the literature. The rarity of osteoma and chondroma of the base of the tongue can also be explained by this theory.

SCIARRETTA.

LARYNX

Bronchogenic Carcinoma with Subcutaneous Metastases.

Charache, Herman, Am. J. Cancer, 37:431, No. 3 (Nov.), 1939.

Clinical and necropsy findings are reported of a case of subcutaneous metastasis from a bronchogenic carcinoma. The incidence in the literature is 2.8 per cent, thirty cases having been recorded. Small nodules usually occur on the same side as the affected bronchus, often quite remote from the primary lesion. Excision of one of these nodules may establish the diagnosis in obscure examples of pulmonary tumors.

JORSTAD.

Laryngocele: Its Comparative Anatomy (Laryngocèle et anatomie comparée).

Rendu, R. (Lyon), Rev. de L. O. R., 60:501 (June), 1939.

By an interesting series of studies of the location of laryngeal sacs in mammals, especially in the anthropoid apes, Rendu concludes that abnormalities of the ventricle of Morgagni may reproduce under vocal stress the various animal types of laryngocele. Rising from the ventricular appendix, which normally extends upward toward the base of the epiglottis, congenital channels may extend backward, upward, or forward. While not more than 100 cases of true laryngocele have been reported, it is interesting that Larrey, Napoleon's surgeon in the Egyptian campaign, first observed them in muezzins calling the faithful to prayer, and observed their likeness to the air sacs of apes.

FENTON.

The Simplification of Technique in Peroral Endoscopy.

Negus, V. E. (London), Brit. Med. J., No. 4120, Page 1223 (Dec. 23), 1939.

The author prefers to carry out esophagoscopy under general anesthesia, namely, intratracheal gas and oxygen. Bronchoscopy is done in adults with sedation followed by local anesthesia, and in children with rectal injection of paraldehyde. This is sometimes supplemented by an inhalation anesthetic.

The patient is held in position by a headrest elaborated from that of Haslinger.

Laryngoscopes with proximal twin lamps which deliver parallel beams of light are used. These beams are oblique and overlap just below the lower aperture of the tube.

Bronchoscopes differ from the Jackson type in having the proximal third funnel-shaped. They are distally lighted.

Esophagoscopes use the oblique lighting and are large, up to a full size of 20 x 18 mm. The inner walls are dull.

DEAN, JR.

TRACHEA

Sarcoma of the Trachea: Report of Two Cases.

Weinberg, Tobias, Am. J. Cancer, 37:201, No. 2 (Oct.), 1939.

Two cases of sarcoma of the trachea are reported with the necropsy findings:

(1) A myxosarcoma, possibly arising in a mixed tumor of the salivary gland type.

(2) A spindle-cell sarcoma.

A short review of the literature, with clinical and necropsy findings of the two cases are recorded. These two cases bring the total recorded to thirty-four. These two were the only ones encountered in 45,000 surgical specimens and 5,500 autopsies at Mt. Sinai Hospital.

Sarcomas of the trachea are of relatively low malignancy, death usually due to mechanical obstruction and subsequent cardiac failure.

JORSTAD.

EAR

Otosclerosis: Clinical and Pathological Findings (Zur Klinik und pathologischen Anatomie der Otosklerose).

Nager, F. R. (Zurich), Acta Oto-Laryng., 27:542, No. 5 (Sept.-Oct.), 1939.

Personal observation of 1,146 cases and the huge series of temporal bones in the Zürich clinic leads Professor Nager to the opinion that 10 per cent of German Swiss people have indications of otosclerotic changes, but that only a few present manifest symptoms.

The sex proportion is 1 male to 1.8 female. More than half the cases presented symptoms between 16 and 30 years of age, and gave a history of hereditary or familial deafness. Unilateral deafness for several years, checked histologically, was found in 10 per cent. Influence of marriage was discounted by the fact that 350 were unwed and 383 married women; in the latter group only 80 got worse with pregnancy.

Though the cause remains unknown, its histogenesis has been clarified. The otosclerotic process exhibits an osteodystrophy limited to the labyrinthine capsule, progressing very slowly and exhibiting every stage of resorption of normal bony structure with metaplasia and substitution of more or less developed bony tissue, first fibrotic, then blue-staining layers, then eosinophilic bony plates changing finally to callous-like and sclerotic regions containing marrow spaces. Rarely invading the semicircular canals, the process only disturbs hearing when the channels of the vestibular and acoustic nerves are invaded, or when the footplate of the stapes is ankylosed. The latter occurred in only 1 per cent; in 15 per cent, lesions were unilateral.

This contribution is brief, authoritative and clear.

FENTON.

Treatment of Otorrhea By Argyrol Displacement.

Reid, W. Ogilvy (Sutton, Surrey), Brit. Med. J., No. 4121, Page 1271 (Dec. 30), 1939.

Cases of chronic otorrhea in which the indications for operation were doubtful were chosen for this treatment. Those cases with a frankly purulent discharge responded better than those with a mucoid discharge.

The ear was cleansed thoroughly, the patient placed in a reclining position with the affected ear uppermost, and the canal filled with 10% argyrol to the lower level of the tragus. A Siegle speculum was fitted into the canal with the bulb compressed, and the bulb allowed to expand. This was done three times.

An alternative measure consisted of closing the canal by pressure over the tragus and making pressure in front of the canal.

The average number of daily treatments necessary to achieve a dry ear was twelve. Of 116 cases, 72 (62.07%) were cured.

DEAN, JR.

Influence of Parathyroidectomy on Otosclerosis (Otosclerose ou otospongiose—influence exercée par la parathyróidectomie sur l'audition).

Alonso, J. M., and Chiarino, A. (Montevideo), Acta Oto-Lar., 27:123, No. 2 (March-April), 1939.

Results since 1935 on 19 cases show marked improvement for the spoken voice and audiometer, especially for the high conversational range, for one to two years following operation. Parathyroidectomy (unilateral) was done in 12, ligation of the branches of the inferior thyroid artery in seven cases. Eleven cases were female; one remained much improved for three years, but lost her hearing with her first pregnancy. Gradual diminution of the original gain occurred in all cases. Hypercalcemia, almost always present, was much lessened after the operative procedures.

FENTON.

Infantile Mastoiditis (La Otomastoiditis del lactante).

Oreggia, J. C. (Montevideo), An. O. R. L. Uruguay, 9:57, No. 2, 1939.

Classifying 49 cases in the past five years, 37 showed external manifestations; of these 15 showed otitis and six did not—so-called "primary" mastoid involvement. Sixteen other otomastoiditis cases were complicated, three by tuberculosis and four by grave digestive disturbances. Among this series seven were fatal.

Twelve latent cases with otoantritis, operated upon because of digestive, athreptic and septicemic symptoms including dehydration and acidosis, showed positive operative results in ten; two were negative; eight fatalities occurred, of which five were gastrointestinal, and three pneumonia and digestive.

The author bases his prognosis upon the child's general appearance, color and skin texture, along with temperature and digestive capabilities, following Marriott, Costen and Carmack. Surgically, he advocates LeMee's two-step procedure; Wilde's incision first, followed according to indication, by removal of necrotic bone and curettage of the antral cells after 48 hours to 10 days. Of course a few cases required complete operation in one stage. This is an excellently documented study with complete clinical details.

FENTON.

MISCELLANEOUS

Anatomical Anomaly of Frontal Sinuses and Its Pathological Importance in a Severe Case of Pansinusitis. (Anomalia anatomica dei seni frontali e sua importanza patogenetica in un caso grave di pansinusite.)

Bombelli, U. (Roma), Valsalva, 14:57-72 (Feb.), 1938.

This is a detailed case report of a right sided pansinusitis in which the infection invaded the left frontal and maxillary sinuses.

The interesting clinical point is the extension of the infection from the right sinuses to the left. Study of the roentgenograms revealed an extremely large right frontal sinus with a recess stretching far over the middle of the left supra-orbital ridge, and a left frontal engulfing this right frontal all the way over to the medial line of the frontal bone. Surgically it was discovered that the intra-sinus septum of the frontal was totally absent. The frontal sinus, which was considered a right frontal, evidently represented both right and left frontal sinuses without a septum, because a distinct nasofrontal duct opened into the right and left meatuses respectively. The left frontal sinus, also had its opening into the middle meatus, and therefore was considered a supernumerary frontal. A small dehiscence existed between the two left frontal sinuses.

The author reports this anomaly, because he was unable to find a single similar clinical report in his bibliographical search.

SCIARRETTA.

Acute Osteomyelitis of the Superior Maxilla in Young Infants.

Asherson, N., J. Laryng. and Otol., 54:691 (Dec.), 1939.

This disease is here described as being primarily in the tooth socket of the upper deciduous unerupted first molar. It may extend to involve the antrum, canine fossa, orbit and palate. The origin in the tooth socket accounts for the appearance of canine fossa swelling before the appearance of nasal discharge. The bacterial agent is a staphylococcus, while acute antrum or ethmoid infections are due to a streptococcus.

The onset is characterized by fever and rapidly developing edema of the lids, proptosis, chemosis of the palpebral and bulbar conjunctiva, and swelling of the cheek. Soon develops a unilateral nasal discharge from which a staphylococcus can be cultured. Swell-

ing develops over the superior alveolus and sometimes over the hard palate. Draining sinuses may appear in the areas of swelling. Constitutional symptoms and signs become severe. The acute stage lasts about ten days and then a chronic stage sets in which is characterized by draining fistulae and sequestra, and may persist for years.

Treatment consists of external drainage wherever pus is formed. Internal drainage has not prevented the formation of external fistulae. The orbital abscess is drained externally and through the middle meatus into the nose, the antrum through the inferior meatus and the canine fossa, or alveolar abscess through the mouth.

In the chronic stage sequestra must be located and removed.

Four cases are recorded in great detail along with five cases of orbital swellings in infants, due to other causes.

DEAN, JR.

HENRY LAWRENCE SWAIN

HENRY LAWRENCE SWAIN

(1864-1940)

Henry L. Swain, for many years a notable figure in American Laryngology, died January 11th at his home in New Haven. He was the son of George Hussey Swain, captain of a clipper ship, and Henrietta Weeks.

The first nine years of Henry's life were spent on the Island of Nantucket, and his early education was received at the famous "Cent School", which was supported by each pupil bringing a cent in his dinner pail every day. It is a tradition in the Swain family that even as a small child Henry had leanings towards the healing art and that he made up sugar water and bread pills and went around dosing his family and friends. When he was nine years of age his father moved to New Haven and Henry's education was continued in the public schools of that city.

At the time Henry Swain entered the Yale Medical School, admission was based on a certificate of graduation from a high school, and this accounts for the fact that when Swain graduated in 1884 he was only twenty years of age and could not therefore legally practice medicine. The old record books which contain his marks are still preserved and show that he was an excellent student. On graduation he decided to go abroad, and in company with his friend and classmate, Oliver T. Osborne, went to Leipzig. Here Henry Swain spent two profitable years, the major part of his work being done under Professor Hagen, who was the Professor of Laryngology and a well-known authority on the subject. Dr. Osborne states that in addition to his work in laryngology, Swain also took courses on diseases of the eye, ear and throat. Swain was a good linguist and before the university opened in the fall he went into the Hartz Mountains and lived with a German family so that he could acquire the language more competently. Professor Hagen made him his assistant. During Swain's second year the Professor fell ill and it devolved upon Swain to carry on a good deal of his work. Swain's "arbeit" under Hagen had to do with the lingual tonsil, and it is stated that Henry Swain was probably the first to describe this structure adequately. According to Wright, this structure had been mentioned by Stöhr and Retterer, but the first definitive description appears to have been Swain's. The article was printed in the Deutsches Archiv für Klinische Medizin, 1886.

On his return from Germany in 1886, Swain opened an office in York Street and here remained for many years, in fact until he removed into the Colonial Building on the corner of Church and Elm Streets, where he spent the remainder of his professional career.

When Dr. Swain returned to New Haven in 1886, he was appointed Lecturer on Diseases of the Throat and Ear in the Yale Medical School, and this position he held until 1895. During 1888 and 1889 he also acted as Demonstrator in Anatomy. In 1895 he was appointed Clinical Professor of Laryngology and Otology. In 1915 his title was changed to Clinical Professor of Otology, and in 1935 he became Emeritus Professor of Otology. He was Consulting Laryngologist to the New Haven Dispensary from 1887 to 1940, and Consulting Laryngologist to the New Haven Hospital from 1893 to 1940.

Dr. Swain was interested not only in hospital and dispensary work and in teaching but also in the local and national medical societies. He was a member of the New Haven Medical Society for many years and was its President quite early in his career. He represented the County Society as its Counselor to the State Society for several years. He was prominent in the affairs of the American Laryngological Association, of which he was President in 1901. In 1939 he was given honorary membership in this Association, being at that time one of four men in the United States on whom this honor had been conferred. He always took an active part in the discussions when subjects in which he was especially interested came up in meetings.

Dr. Swain was interested in tuberculosis, and his name appears among the original incorporators of the Gaylord Farm Sanatorium. He contributed to medical literature from time to time. There were certain subjects in which he was particularly interested, notably bronchial asthma and the diseases of the adenoid tissue in the nasopharynx and pharynx. He was probably the first to use suprarenal extract in nose and throat work, and his paper on "The Local Use of the Aqueous Extract of the Suprarenal Glands of the Sheep in the Nose and Throat" appeared in the New York Medical Journal for 1898. Most of his papers on asthma, its relation to nasal disease, its origin, and particularly its surgical treatment, appear between 1892 and 1900.

Dr. Swain was the first man, in all probability, who removed adenoids and tonsils in Connecticut; certainly one of the first in New England.

It is of course difficult to appraise at such close range the achievements of any man. There can be no doubt, however, that Dr. Swain was one of the outstanding American laryngologists of his time. He had excellent training, he was a student, he had good judgment, and his contributions to the subject showed evidence of an alert and active mind constantly on the lookout for new developments. His personality was distinguished and pleasing. His patients became sincerely attached to him, and his death is a loss both to his patients and to his confereres.

DOCTOR HEINRICH V. NEUMANN

(1873-1939)

Doctor Heinrich V. Neumann was born in Hungary in 1873. He received his medical degree from the University of Vienna in 1898. Under the inspiring influence of Politzer (otology) and Weichselbaum (pathological anatomy), Dr. Neumann's outstanding ability soon asserted itself. He steadily advanced from one to another of the following positions: 1907, Dozent in Otology, Vienna University; 1911, Professor of Otology, University of Vienna; 1914, Chief of Otological Department, Allgemeines Krankenhaus in Vienna; 1933, Chief of Laryngological Department (formerly Hajek Service).

In 1912 he visited a number of cities in this country during the meeting of the International Otological Congress, and later gave courses in otology. Signal honors and distinctions were bestowed upon him in Europe, among them, The Royal Society of Medicine, London; Scottish Otological Society; Collegium Amicitiae Sacrum, French, Hungarian, Rumanian, and some other otological societies. As a teacher of otology and as a clinician his outstanding ability received world-wide recognition. His investigations have been noteworthy and his contributions to the literature of his branch of medicine, while not voluminous, have been of high and permanent value. For a generation his name has been associated with an operative procedure for labyrinthine suppuration with a complicating meningitis.

A genial personality, a master in his special field who, for many years as an inspiring teacher of men throughout the world, advanced clinical otology to its present high state; sought for because of his professional standing by crowned heads of Europe and many other distinguished individuals; a humanitarian in the true sense because of his devotion to the care of the poor—these are some of the outstanding qualities and attributes which characterized this renowned otologist. Dr. Neumann died in New York on November 5, 1939, at the age of 66, after an illness of two months.

J. L. M.

PUBLICATIONS OF DOCTOR HENRICH V. NEUMANN

1. Der otitische Kleinhirnabszesz. Wien., 1907.

2. Zur Bakteriologie der akuten Mittelohreiterungen. Verhandl. d. dtsch. otol. Ges., 1907, S. 95; with E. Ruttin: Zur Aetiologie der akuten Otitis. Arch. f. Ohren- u.s.w. Heilk., 79:1, 1909.

3. Zur Klinik und Therapie der otogenen Bulbusthrombose der Vena Jugularis. Mitt. a.d. Grenzgez. der Medizin und Chir., 31, 1918.

4. Nystagmus and Its Clinical Significance. Laryngoscope, 1921.

5. Der otitische Hirnabszesz, Jahreskurse f. aerztl. Fortbildung (Nov.), 1926.

6. Indikation und Technik der Fruehoperation der akuten Mastoiditis. Zeitschr. f. Hals- u.s.w. Heilk., 15:273, 1926; with F. Fremel: Die Physiologie der Zentren und Bahnen dez Vestibularapparates in Handb. d. Hals- u.s.w. Heilk. by Denker und Kahler, 1926.

7. Die conservative Radikaloperation der chronischen Mittelohrentzuendungen. Acta oto-laryng. (supp. 7), 121, 1928.

8. Chirurgie d. Ohres in Lehrbuch d. Chirurgie by A. Eiselsberg, Vienna, 1930.

9. Zur Klinik und Pathologie der Otitis media acuta fibrinosa. Zeitschr. f. Hals- u.s.w. Heilk., 31:410, 1932.

10. Zur Pathologie und Therapie der otogenen Meningitis. Acta oto-lar., 20:102, 1934.

11. Neumann's Operation for Labyrinthine Suppuration: Schlander, E., und Beck, O., Technik der Neumannschen Labyrinthoperation. Ztschr. f. Hals- u.s.w. Heilk., 19:59, 1927.

Books Received

Medicine of the Ear.

Prepared under the Editorship of Dr. Edmund Prince Fowler, Jr., Assistant Clinical Professor of Otolaryngology, College of Physicians and Surgeons, Columbia University; and Assistant Surgeon, Manhattan Eye, Ear and Throat Hospital, New York. With a Foreword by John Devereux Kernan, M.D., Professor of Otolaryngology, College of Physicians and Surgeons, Columbia University. Cloth. Royal 8 vo. of 603 pages. Thomas Nelson and Sons, New York, 1939

A companion volume to the recently published Looseleaf Surgery of the Ear, this is in effect a series of 17 monographs by a group of distinguished otologists, with two exceptions American. The material though presented in textbook fashion, actually covers a field more comprehensive than is attempted in the average textbook. It is well documented and illustrated, and the personnel of contributors assures its authenticity in so far as any work dealing with the rapidly advancing science of the ear may be said, at any time, to be authentic.

The publishers have recognized the rapid growth and development of the subject by presenting their volume in looseleaf form. This book is highly recommended to the graduate student and the practicing otologist.

Epidemic Encephalitis: Etiology, Epidemiology, Treatment.

Third Report by the Matheson Commission; Willard C. Rappleye, Chairman. 16 mo. of xi + 493 pages with numerous charts. Columbia University Press, New York, 1939. Price, $3.00.

"The Matheson Commission for the Study of Epidemic Encephalitis has continued its investigations in the direction outlined in earlier reports. The long continued follow-up of a large number of patients suffering from this disease or other conditions closely simulating it has provided a volume of clinical evidence and experience that has proven of greatest value in differential diagnosis and in the appraisal of methods of treatment."

Two hundred and ninety-eight pages are devoted to the bibliography of the subject from the beginning of 1930 to the first half of 1938.

The Surgery of Injury and Plastic Repair.

By Samuel Fomon, Ph.D., M.D., Formerly Major Medical Corps, U. S. Army. ix + 1409 pages profusely illustrated. Many diagrams in color. The Williams and Wilkins Company, Baltimore, 1939. Price, $15.00.

The rhinologist who practices this type of surgery will find much to interest him in this volume. The first five hundred pages are devoted to the underlying principles of plastic surgery, and such subjects as fluid, salt and acid base balance, shock and anesthesia. Almost six hundred more pages are given over to the plastic surgery of the nose, the auricle, the maxillo-facial region, the lip, cleft lip and cleft palate, and the mandible.

The material is presented with great completeness in the impersonal style of the textbook. The illustrations so essential to the satisfactory demonstration of this subject are simple and well executed. Color is used to excellent advantage in many of the diagrams.

An altogether satisfying book.

Notice

Fourth Annual Graduate Course in Otolaryngology

The Fourth Annual Graduate Course in Otolaryngology will be conducted at the Cincinnati University College of Medicine during the week of May 20-25, 1940. The Course consists chiefly of intensive operative work on the cadaver and is conducted by the Faculty of the Medical College and the Staff of the Cincinnati General Hospital. The course is limited to twenty-five practicing otolaryngologists.

Samuel Iglauer, M.D.

Contents.

CONTENTS—Continued